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ABSTRACT

This report focuses on the evidence about the health consequences of smoking for women, and is intended to serve the public health and medical communities as a unified source of existing scientific research. The major issues about tobacco use and women's health are examined, including trends in consumption, biomedical evidence, and determinants of smoking initiation, maintenance, and cessation. The biomedical aspects of smoking are presented in terms of mortality, morbidity, cardiovascular diseases, cancer, pregnancy and infant health, peptic ulcer disease, drug reactions and interactions, food constituents, and responses to diagnostic tests. The psychosocial and behavioral aspects of smoking in women are discussed, along with the documented increases in the risk of contracting lung cancer, heart disease, or lung disease. The harmful effects of pregnant mothers' smoking behaviors are also documented. Additionally, recent data are enumerated to indicate a trend of decreasing tobacco usage among women in response to the warnings of the government, voluntary agencies, and physicians. (Author/NRB)

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THE HEALTH CONSEQUENCES OF SMOKING FOR WOMEN

a report of the Surgeon General 1979

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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Office of the Assistant Secretary for Health
Office on Smoking and Health

THE SECRETARY OF HEALTH, EDUCATION, AND WELFARE
WASHINGTON, D.C. 20201

The Honorable Thomas P. O'Neill,Jr.
Speaker of the House of Representatives
Washington, D.C. 20515

Dear Mr. Speaker:

I hereby submit the 12th annual report that the Department of Health, Education, and Welfare (DHEW) has prepared for Congress as required by the Public Health Cigarette Smoking Act of 1969, Public Law 91-222, and its predecessor, the Federal Cigarette Labeling and Advertising Act. This report is one of the most alarming in the series.

It clearly establishes that women smokers face the same risks as men smokers of lung cancer, heart disease, lung disease and other consequences. Perhaps more disheartening is the harm which mothers' smoking causes to their unborn babies and infants.

The report is not all bad news. It presents recent data showing that women are turning away from smoking in response to the warnings of government, voluntary agencies and physicians. The precipitate rise in women's deaths from lung cancer and chronic lung disease demand that this trend away from cigarettes be accelerated. Our scientists expect that by 1983, the lung cancer death rate will exceed that of any other type of cancer among women.

Citizens of our free society may decide for themselves whether to smoke cigarettes. The health consequences of this decision make it imperative for their government to assure that the decision is an informed one. This series of reports is one way in which DHEW is striving to meet this critical responsibility.

Sincerely yours,

Patricia Roberts Harris

PREFACE

This report is more than a factual review of the health consequences of smoking for women. It is a document which challenges our society and, in particular, our medical and public health communities.

This report points out that the first signs of an epidemic of smoking-related disease among women are now appearing. Because women's cigarette use did not become widespread until the onset of World War II, those women with the greatest intensity of smoking are now only in their thirties, forties, and fifties. As these women grow older, and continue to smoke, their burden of smoking-related disease will grow larger. Cigarette smoking now contributes to one-fifth of the newly diagnosed cases of cancer and one-quarter of all cancer deaths among women—more cancer and more cancer deaths among women than can be attributed to any other known agent. Within three years, the lung cancer death rate is expected to surpass that for breast cancer. A similar epidemic of chronic obstructive lung disease among women has also begun.

Four main themes emerge from this report to guide future public health efforts.

First, women are not immune to the damaging effects of smoking already documented for men. The apparently lower susceptibility to smoking-related diseases among women smokers is an illusion reflecting the fact that women lagged one-quarter century behind men in their widespread use of cigarettes.

Second, cigarette smoking is a major threat to the outcome of pregnancy and well-being of the newborn baby.

Third, women may not start smoking, continue to smoke, quit smoking, or fail to quit smoking for precisely the same reasons as men. Unless future research clarifies these differences, we will find it difficult to prevent initiation or to promote cessation of cigarette smoking among women.

Fourth, the reduction of cigarette smoking is the keystone in our nation's long term strategy to promote a healthy lifestyle for women and men of all races and ethnic groups.

The Fallacy of Women's Immunity

All of the major prospective studies of smoking and mortality have reached consistent conclusions. Death rates from coronary heart disease, chronic lung disease, lung cancer, and overall mortality rates are significantly increased among both women and men smokers. These risks increase with the amount smoked, duration of smoking, depth of inhalation, and the "tar" and nicotine delivery of the cigarette smoked.

In these studies, conducted during the past three decades, relative mortality risks among female smokers appeared to be less than those of male smokers. It is now clear, however, that these studies were comparing the death rates of a generation of established, lifelong male smokers with a generation of women who had not yet taken up smoking with full intensity. Even those older women who reported smoking a large number of cigarettes per day had not smoked cigarettes in the same way as their male counterparts. Now that the cigarette smoking characteristics of women and men are becoming increasingly similar, their relative risks of smoking-related illness will become increasingly similar.

This fallacy of women's apparent immunity is clearly illustrated by differences in the timing of the growth in lung cancer among men and women in this century. Lung cancer deaths among males began to increase during the 1930s, as those men who had converted from other forms of tobacco to cigarette smoking before the turn of the century gradually accumulated decades of inhaled tobacco exposure. By the time of the first retrospective studies of smoking and lung cancer in 1950, two entire generations of men had already become lifelong cigarette smokers. Relatively few women from these generations smoked cigarettes, and even fewer had smoked cigarettes since their adolescence. Those young women who had taken up smoking intensively during World War II were only in their twenties and thirties. In 1950, women accounted for less than one in twelve deaths from lung cancer.

Thereafter, the age adjusted lung cancer death rate among women accelerated, and the male predominance in lung cancer declined. Lung cancer surpassed uterine cervical cancer as a cause of death in women. By 1968, as the findings of many large population prospective studies were being published, women accounted for one-sixth of all lung cancer deaths. These studies found that women cigarette smokers had 2.5 to 5 times greater death rates from lung cancer than women nonsmokers. By 1979, women accounted for fully one-fourth of all lung cancer deaths. Over the next few years, women cigarette smokers' risk of lung cancer death will approach 8 to 12 times that of women nonsmokers, the same relative risk as that of men.

Lung cancer has four main histological types: epidermoid, small cell, adenocarcinoma, and large cell carcinoma, As several studies have shown, the incidence of each of these types of lung cancer displays a clear relationship to cigarette smoking among both men and women. Epidermoid and small cell lung cancer appear to be more prominent among men, while adenocar-

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cinoma of the lung now appears to be more prominent among women.

The recent acceleration of lung cancer incidence among women has in fact been more rapid than the corresponding growth of lung cancer among men in the 1930s. Again, this difference in the initial rate of acceleration of lung cancer incidence does not refute the demonstrated causal relation between cigarette smoking and lung cancer among both sexes. Instead, differences in the rate of increase of lung cancer incidence may reflect changes in the carcinogenic properties of cigarette smoke, the style of cigarette smoking, or the interaction of cigarette smoking with other environmental hazards. It is noteworthy that those men who died of lung cancer in the 1930s came from a generation that had gradually converted to cigarettes from other, non-inhaled forms of tobacco. By contrast, the first regular tobacco users among women were almost exclusively cigarette smokers.

The 1979 Report on Smoking and Health documented numerous instances where cigarette smoking adds to the hazards of the workplace environment among men. Among women, this report reveals two such occupational exposures—asbestos and cotton dust—which have been clearly demonstrated to interact with cigarette smoking. The fact that evidence is limited among women does not imply that women are protected from the dangerous interactions of smoking and occupational exposures.

Pregnancy, Infant Health, and Reproduction

Scientific studies encompassing various races and ethnic groups, cultures and countries, involving hundreds of thousands of pregnancies, have shown that cigarette smoking during pregnancy significantly affects the unborn fetus and the newborn baby. These damaging effects have been repeatedly shown to operate independently of all other factors that influence the outcome of pregnancy. The effects are increased by heavier smoking and are reduced if a woman stops smoking during pregnancy.

Numerous toxic substances in cigarette smoke, such as nicotine and hydrogen cyanide, cross the placenta to affect the fetus directly. The carbon monoxide from cigarette smoke is transported into the fetal blood and deprives the growing baby of oxygen. Fetal growth is directly retarded. The resulting reduction in fetal weight and size has many unfortunate consequences. Women who smoke cigarettes during pregnancy have more spontaneous abortions, and a greater incidence of bleeding during pregnancy, premature and prolonged rupture of amniotic membranes, abruptio placentae and placenta previa. Women who smoke cigarettes during pregnancy have more fetal and neonatal deaths than nonsmoking pregnant women. A relation between maternal smoking and Sudden Infant Death Syndrome has now been established.

The direct harmful effects of smoking on the fetus have long term consequences. Children of mothers who smoked during pregnancy lag measurably in physical growth; there may also be effects on behavior and cognitive development. The extent of these deficiencies increases with the number of cigarettes smoked.

The damaging effects of maternal smoking on infants are not restricted to pregnancy. Nicotine, a known poison, is found in the breast milk of smoking mothers. Children whose parents smoke cigarettes have more respiratory infections and more hospitalizations in the first year of life.

Women who smoke cigarettes have more than three times the risk of dying of stroke due to subarachnoid hemorrhage, and as much as two times the risk of dying of heart attack in comparison to nonsmoking women. The use of oral contraceptives in addition to smoking, however, causes a markedly increased risk, including a 22-fold increase in the risk of subarachnoid hemorrhagic stroke and a 20-fold increase in heart attack in heavy smokers.

Why Do Women Smoke?

Cigarette consumption in this country is now declining. Annual per capita consumption has decreased from 4,258 in 1965 to an estimated 3,900 in 1979. From 1965 to 1979, the proportion of adult male cigarette smokers declined from 51 to 37 percent. Not only have millions of men quit smoking, but the rate of initiation of smoking among adolescent males has now slowed.

From 1965 to 1976, the proportion of adult women cigarette smokers remained virtually unchanged at 32 to 33 percent. Since 1976, however, the proportion of adult women cigarette smokers appears to have declined to 28 percent. Although adult women are now beginning to quit smoking at rates comparable to adult men, the rate of initiation of smoking among younger women has not declined.

This report documents numerous differences by sex in the perceived role of cigarette smoking, in attitudes toward health and lifestyle, and in methods of coping with stress, anger, and boredom. Yet the significance of these differences, and their relation to differences in smoking patterns, remains poorly understood.

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Although it is frequently observed that women in organized smoking cessation programs have more severe withdrawal symptoms and lower rates of successful quitting than men, these observations have not been systematically confirmed for the general population. In the past, women may have attempted to quit or succeeded in quitting smoking less frequently than men. The recent decline in the proportion of women smokers, however, suggests that women's attempted and successful quitting rates have now increased.

Although weight gain is a frequently cited consequence of quitting smoking, the association of weight gain with cessation of smoking has not been the subject of sufficient scrutiny. Controlled studies with careful measurement on representative populations of women do not exist. The impact of the fear of weight gain after quitting has not been adequately examined. If weight gain does result from cessation of smoking, its exact mechanism must be determined.

Even more problematic are marked differences by sex in the distribution of smoking prevalence by occupation. Men with advanced education and professional occupations have taken the lead in quitting smoking, but women in administrative and managerial positions have relatively high smoking prevalence rates. Although 20 percent or fewer male physicians smoke, the proportions of cigarette smokers among women health professionals, especially nurses and psychologists, remain disturbingly high.

Recent changes in smoking prevalence among black women and men have paralleled those of the general population. From 1965 to 1979, the proportion of black women cigarette smokers declined from 34 to 29 percent, while the proportion of black men smokers declined from 61 to 42 percent. However, differences by race in the onset, maintenance, and cessation of smoking have not been adequately explored. Little is known about cigarette smoking among other ethnic and minority groups.

Adolescent Smoking

The health consequences of smoking evolve over a lifetime. Evidence continues to accumulate, for example, that cigarette smoking produces measurable lung changes in adolescence and young adulthood. Young cigarette smokers of both sexes show more evidence of small airway dysfunction, and a higher prevalence of cough, wheezing, phlegm production, and other respiratory symptoms. The health damage due to cigarette smoking increases when an individual begins regular smoking earlier in life. Yet, as this report documents, the average age of onset of regular smoking among women has continuously declined during the last 50 years, and continues to decline.

According to a recent survey by the National Institute of Education, cigarette smoking among adolescent girls now exceeds that among adolescent boys. In the 17–19 year age group, there are almost 5 female cigarette smokers for every 4 male cigarette smokers. The causes of this inversion are far from clear. We do not yet understand the signal events in the initiation of smoking among young women. It is possible that parents set examples concerning lifestyle, health attitude, and risk-taking much earlier in childhood. The beginning of junior high school or entrance into the work force may be equally critical events. We do not know enough about an adolescent's sense of competence and self-mastery, and how these roles differ among women and men. Although smoking patterns among girls correlate with parental, peer and sibling smoking habits, educational level, type of school curriculum, academic performance, socioeconomic status, and other forms of substance abuse, the practical significance of these empirical correlations is unclear.

Women and the Changing Cigarette

As this report documents, the proportion of men and women smokers using brands with lowered "tar" and nicotine continues to grow. Adolescents of both sexes have followed this trend, to the point where nonfilter cigarettes are relatively rare among young adults.

Although the preponderance of scientific evidence continues to suggest that cigarettes with lower "tar" and nicotine are less hazardous, four serious warnings are in order.

First, the reported "tar" and nicotine deliveries of cigarettes are standardized machine measurements. They do not necessarily represent the smoker's actual intake of these substances. Evidence is now mounting that individuals who switch to cigarettes with lowered "tar" and nicotine inhale more deeply, smoke a greater proportion of their cigarettes, and in some cases smoke more cigarettes.

Second, "tar" and nicotine are not the only dangerous chemical components of cigarette smoke. Many conventional filter cigarettes, in fact, may deliver more carbon monoxide than nonfilter cigarettes.

Third, it has not been established that lower "tar" and nicotine cigarettes have less harmful effects on the unborn fetus and baby; on women and men at high risk for developing coronary heart disease, such as those with elevated cholesterol or high blood pressure; or on workers with adverse occupational

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exposures. It has not been established that switching to a lower "tar" and nicotine cigarette has any salutary effect on individuals who already have smoking-related illnesses, such as coronary heart disease, chronic bronchitis, and emphysema.

Fourth, even the lowest yield cigarettes present health hazards for both women and men that are very much higher than smoking no cigarettes at all.

The single most effective way for both women and men smokers to reduce the hazards associated with cigarettes is to quit smoking.

As this report demonstrates, little is known about the effects of these product changes on the initiation, maintenance and cessation of smoking, particularly among women. It has not been determined whether the availability of cigarettes with lowered "tar" and nicotine has made it easier for young women to experiment with and become addicted to cigarettes. It is not known whether smokers of the lowest yield cigarettes are more or less likely to attempt to quit, or to succeed in quitting, than smokers of conventional filtertip or nonfilter cigarettes. The extent to which the act of switching to a lower "tar" cigarette serves as a substitute for quitting may differ among women and men.

Public Health Responsibilities

This report, which includes data compiled by individuals from both inside and outside the Government, has confirmed in every way the judgement of the World Health Organization that there can no longer be any doubt among informed people that cigarette smoking is a major and removable cause of ill health and premature death.

Each individual woman must make her own decision about this significant health issue. Secretary Harris has noted that the role of the Government, and all responsible health professionals, is to assure that this decision is an informed one. In issuing this report, we hope to help the public health community accomplish this purpose.

Julius B. Richmond, M.D.
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INTRODUCTION AND SUMMARY.

INTRODUCTION AND SUMMARY

The 1980 Report on the Health Consequences of Smoking focuses upon the evidence relating cigarette smoking to health effects in women. It is not presented as a detailed discussion of the entire range of effects of smoking on health. Such a detailed review of all existing evidence can be found in the 1979 Report of the Surgeon General on Smoking and Health. Instead, this volume on smoking and women's health is offered as a review and reappraisal of smoking and major health relationships specifically in women. It is intended to serve the medical community as a unified source of existing scientific evidence about health effects of smoking cigarettes for women. As an examination of current knowledge, it will logically lend itself to application in both the personal and public health arenas.

Its content is the work of numerous scientists within the Department of Health, Education, and Welfare, as well as scientific experts outside that organization.

This volume examines the major issues relating tobacco use to women's health including trends in consumption, the biomedical evidence of the health effects of cigarette usage by women, and determinants of smoking initiation, maintenance, and cessation.

This section summarizes the principal findings of this report. It is hoped that the entire volume will serve to highlight the established risks of smoking for women and their children, as well as to define the areas in need of further investigation.

Patterns of Cigarette Smoking

1. Women have differed from men in their historical onset of widespread cigarette use, in the rate of diffusion of smoking among each new birth cohort, in their intensity of cigarette smoking and their use of various types of cigarettes.

2. Men took up cigarette smoking rapidly at the beginning of the twentieth century, especially during World War I. Cigarettes rapidly replaced other forms of tobacco.

By 1925, approximately 50 percent of adult males were cigarette smokers. Smoking among men accelerated rapidly during World War II. By 1950, the prevalence of cigarette use among men approached 70 percent in some urban areas.

3. The onset of widespread cigarette use among women lagged behind that of men by 25 to 30 years. The proportion of adult women smoking cigarettes did not exceed one-quarter until the onset of World War II.

4. Between 1951 and 1963, increasing proportions of women

and men smokers converted to filtertip cigarettes. By 1964, 79 percent of adult women smokers and 54 percent of adult men smokers used filter cigarettes.

5. After reaching a peak value of 4,336 in 1963, annual per capita consumption of cigarettes declined in 1964, 1968–70, and in the period since 1975. The most recent estimate of 3,900 cigarettes per capita in 1979 is approximately equal to that observed in 1952.

6. From 1965 to 1978, the proportion of adult men cigarette smokers declined from 51 to 37 percent. The preliminary estimate of adult men's smoking prevalence for 1979 is 36.9 percent. From 1965 to 1976, the proportion of adult women smokers remained virtually unchanged at 32 to 33 percent. Since 1976, the proportion of women smokers has declined to below 30 percent. For 1979, the preliminary estimate of adult women's smoking prevalence is 28.2 percent. The overall smoking prevalence of 32.3 percent for both sexes in 1979 represents the lowest recorded value in at least 45 years.

7. The proportion of adult smokers attempting to quit smoking declined from 1970 to 1975, but increased in 1978–1979. In contrast to past years, the proportions of women and men now attempting to quit smoking, and their reported quitting rates, are indistinguishable. Approximately one in three adult smokers now makes a serious attempt to quit smoking during the course of a year. Approximately one in five of those who attempt to quit subsequently succeed.

8. The proportion of adult smokers using lower "tar" and nicotine brands has increased substantially. In 1979, 39 percent of adult women smokers and 28 percent of adult men smokers reported primary brands with F.T.C. "tar" delivery less than 15.0 milligrams. It is not known whether smokers of the lowest "tar" cigarettes are more or less likely to attempt to quit smoking, or to succeed in quitting, than smokers of conventional filtertip or non-filter cigarettes.

9. The average number of cigarettes smoked by women and men current smokers has increased. The relationship of this finding to recent declines in the average F.T.C. "tar" and nicotine deliveries of cigarettes is not well understood.

10. With each successive generation, the smoking characteristics of women and men have become increasingly similar.

11. Among women, the average age of onset of regular smoking progressively declined with each successive birth cohort—from 35 years of age for those born before 1900, to 16 years of age among those born 1951 to 1960. The average age of onset of regular smoking among young women is now virtually identical to that of young men.

12. Maximum smoking prevalence rates have declined substantially in recent birth cohorts of men. Men born 1931 to 1940 reached a peak smoking proportion of 61 percent during 1960–62, while men born 1941 to 1950 reached a peak smoking proportion of 58 percent in 1968–69. Men born 1951 to 1960 reached a peak smoking proportion of 40 percent in 1976. Among recent cohorts of women, peak smoking prevalence rates have declined to a much smaller extent. Women born 1931 to 1940 reached a peak smoking proportion of 45 percent in 1966–68, while women born 1941 to 1950 reached a peak smoking proportion of 41 percent in 1970–73. Women born 1951 to 1960 reached a peak smoking proportion of 38 percent in 1976. Among the generation born 1951 to 1960, the porportions of women and men smoking cigarettes are now virtually identical.

13. The proportions of women and men smokers in each age group have declined. Among those born before 1951, this decline in smoking prevalence resulted mainly from smoking cessation. By contrast, the observed decline in smoking prevalence among younger men born 1951 to 1960 has resulted from both smoking cessation and a lower rate of smoking initiation. This decline in the rate of onset of smoking among young men has not been observed for young women.

14. Recent survey data on adolescent smoking habits reveal that by ages 17 to 19, smoking prevalence among women exceeds that of men. This finding supports the conclusion that the rate of initiation of smoking among young men—but not that of young women—is declining. The future cigarette use of the youngest generations of women is uncertain.

15. With each successive birth cohort, the accumulated years of cigarette smoking per woman has progressively approached the accumulated years of cigarette smoking per man. Each successive birth cohort has also experienced progressively smaller sex differences in the fraction of lifetime years of smoking that represents filtertip cigarette use.

16. Among men born during this century, each successive birth cohort has thus far experienced fewer cumulative years of cigarette smoking, higher proportionate exposure to filtertip cigarettes, and lower smoking prevalence rates. This relationship between birth date and cigarette smoke exposure does not hold for women. Women born 1921 to 1940 have experienced substantially higher smoking prevalence rates than earlier generations. Unless they quit smoking in substantial numbers, these women, currently aged 40 to 59, will surpass older women in total years of cigarette smoking per capita, the total years of nonfilter cigarette smoking per capita, and in the total number of cigarettes smoked. The health consequences of this enhanced

exposure to cigarette smoke among women are likely to be more prominent in the coming decades.

Mortality

1. The mortality ratio for women who smoke cigarettes is about 1.2 or 1.3.

2. Mortality ratios for women increase with the amount smoked. In the largest prospective study the mortality ratio was 1.63 for the two-pack-a-day smoker as compared to nonsmokers.

3. Mortality ratios are generally proportional to the duration of cigarette smoking; the longer a woman smokes, the greater the excess risk of dying.

4. Mortality ratios tend to be higher for those women who begin smoking at a young age as compared to those who begin smoking later.

5. Mortality ratios are higher for those women who report they inhale smoke than for those who do not inhale.

6. Mortality ratios for women tend to increase with the tar and nicotine content of the cigarette.

7. Mortality ratios for female smokers are somewhat less than for male smokers. This may reflect differences in exposure to cigarette smoke, such as starting smoking later, smoking cigarettes with lower "tar" and nicotine content, and smoking fewer cigarettes per day than men.

8. Women demonstrate the same dose-response relationships with cigarette smoking as men. An increase in mortality occurs with an increase in number of cigarettes smoked per day, an earlier age of beginning cigarette smoking, a longer duration of smoking, inhalation of cigarette smoke, and a higher tar and nicotine content of the cigarette. Women who have smoking characteristics similar to men may experience mortality rates similar to men.

Morbidity

The 1979 Report of the Surgeon General summarized the information on smoking and morbidity as follows:

1. In general, female current cigarette smokers report more acute and chronic conditions including chronic bronchitis and/or emphysema, chronic sinusitis, peptic ulcer disease, and arteriosclerotic heart disease, than women who never smoked.

2. There is a dose-response relationship between the number of cigarettes smoked per day and the frequency of reporting for most of the chronic conditions.

3. The age-adjusted incidence of acute conditions (e.g., influenza) for women smokers is 20 percent higher for women who had ever smoked than for nonsmokers.

Additional data from the Health Interview Survey (HIS) is presented:

1. Currently employed women who smoke cigarettes report more days lost from work due to illness and injury than working women who do not smoke.

2. Limitation of activity is reported more commonly among women under the age of 65 who have ever smoked than among those who never smoked.

Cardiovascular Diseases

Coronary heart disease is the major cause of death among both males and females in the U.S. population. The 1979 Surgeon General's Report clearly demonstrated the close association of cigarette smoking and increased coronary heart disease among males. This report reviews the evidence associating cigarette smoking and cardiovascular disease in women:

1. Coronary heart disease, including acute myocardial infarction and chronic ischemic heart disease, occurs more frequently in women who smoke. In general, cigarette smoking increases the risk by a factor of about two, and in younger women cigarette smoking may increase the risk several fold.

2. Cigarette smoking is a major independent risk factor for coronary heart disease in women; it also acts synergistically with other coronary heart disease risk factors producing a risk greater than the sum of the individual risks.

3. The use of oral contraceptives by women cigarette smokers increases the risk of a myocardial infarction by a factor of approximately ten.

4. Women who smoke low "tar" and nicotine cigarettes experience less risk for coronary heart disease than women who smoke high "tar" and nicotine cigarettes, but their risk is still considerably greater than that of nonsmokers.

5. Increased levels of high-density lipoprotein (HDL) are correlated with a reduced risk for an acute myocardial infarction; women cigarette smokers have decreased levels of HDL.

6. Cigarette smoking is a major, independent risk factor for the development of arteriosclerotic peripheral vascular disease in women. Smoking cessation improves the prognosis of the disorder and has a favorable impact on vascular patency following reconstructive surgery.

7. Women cigarette smokers experience an increased risk for subarachnoid hemorrhage; the use of both cigarettes and oral

contraceptives appears to synergistically increase the risk for subarachnoid hemorrhage.

8. Women who smoke cigarettes may be more likely to develop severe or malignant hypertension than nonsmoking women.

Cancer

1. Cigarette smoking is causally associated with cancer of the lung, larynx, oral cavity, and esophagus in women as well as in men; it is also associated with kidney cancer in women.

2. Cigarette smoking accounts for 18 percent of all cancers newly diagnosed and 25 percent of all cancer deaths in women. In 1980, 26,500 of the estimated 101,000 deaths, or over one-quarter of the deaths expected from lung cancer, will occur in women.

3. Women cigarette smokers have been reported to have between 2.5 and 5 times greater likelihood of developing lung cancer than nonsmoking women.

4. Among women the risk of developing lung cancer increases with increasing number of cigarettes smoked per day, duration of the smoking habit, depth of inhalation, and tar and nicotine content of the cigarette smoked. The risk is inversely related to the age at which smoking began.

5. A dose-response relationship has been demonstrated between cigarette smoking and cancer of the lung, larynx, oral cavity, and urinary bladder in women.

6. The rise in lung cancer death rates is currently much steeper in women than in men. It is projected that the age adjusted lung cancer death rate will surpass that of breast cancer in the early 1980s.

7. The rapid increase in lung cancer rates in women is similar to but steeper than the rise seen in men approximately 25 years earlier. This probably reflects the fact that women first began to smoke in large numbers 25–30 years after the increase in cigarette smoking among men. Thus, neither men nor women are protected from developing lung cancer caused by cigarette smoking.

8. Cigarette smoking has been causally related to all four of the major histologic types of lung cancer in both women and men, including epidermoid, small cell, large cell and adenocarcinoma.

9. The use of filter cigarettes and cigarettes with lower levels of "tar" and nicotine by women is correlated with a lower risk of cancer of the lung and larynx compared to the use of high-"tar" and-nicotine or unfiltered cigarettes. The risk posed by smoking

low-"tar" cigarettes, however, is clearly greater than that among females who never smoked.

10. After cessation of cigarette smoking, a woman's risk of developing lung and laryngeal cancer has been shown to drop slowly, equalling that of nonsmokers after 10–15 years.

11. Excessive ingestion of alcohol acts synergistically with cigarette smoking to increase the incidence of oral and laryngeal cancer in women.

Non-Neoplastic Bronchopulmonary Diseases

1. Recent statistics indicate a rising death rate due to chronic obstructive lung disease (COLD) among women. The data available demonstrate an excess risk of death from COLD among smoking women over that of nonsmoking women. This excess risk is much greater for heavy smokers than for light smokers.
2. Women's total risk of COLD appears to be somewhat lower than men's, a difference which may be due to differences in prior smoking habits.
3. The prevalence of chronic bronchitis varies directly with cigarette smoking, increasing with the number of cigarettes smoked per day.
4. There is conflicting evidence regarding differences in the prevalence of chronic bronchitis in women and men. Several recent studies suggest that there is no significant difference in the prevalence of chronic bronchitis between male and female smokers. This may be the result, however, of increasingly similar smoking behavior of women and men.
5. The presence of emphysema at autopsy exhibits a dose-response relationship with cigarette smoking during life.
6. There is a close relationship between cigarette smoking and chronic cough or chronic sputum production in women, which increases with total pack-years smoked.
7. Women current smokers have poorer pulmonary function by spirometric testing than do female ex-smokers or nonsmokers, a relationship which is dose-related to the number of cigarettes smoked.

Interaction Between Smoking and Occupational Exposures

1. The 1979 Surgeon General's Report identified the ways in which smoking cigarettes may interact with the occupational environment. They include:
   a) Facilitation of absorption of physical contamination of cigarettes,
   b) Transformation of workplace chemicals into more toxic substances,

c) Addition of the exposure to a toxic constituent of tobacco smoke to a concurrent exposure to the same constituent present in the workplace,

d) Addition of a health effect due to environmental exposure to a similar health effect due to smoking,

e) Synergy of exposures, and

f) Causation of accidents.

2. Women are entering occupational environments with greater frequency, and thus may be experiencing greater exposures to physical and chemical agents.

3. Cohorts of women with a greater prevalence of smoking are currently reaching the ages of maximal disease occurrence, replacing earlier cohorts with lower cigarette exposures.

4. Physiologic differences in hormonal status between males and females constitute a potential source of differing responses.

5. In the workplace women who are pregnant present a nine-month exposure opportunity, including potential teratogenic and perinatal mortality effects.

6. Concurrent exposure of women to smoking and asbestos resulted in a clear excess of cancer of the lung.

7. Women smokers exposed to cotton dust run a higher risk of developing byssinosis, bronchitic syndromes, and abnormal pulmonary function tests than nonsmoking women.

Pregnancy and Infant Health

1. Babies born to women who smoke during pregnancy are, on the average, 200 grams lighter than babies born to comparable nonsmoking women.

2. The relationship between maternal smoking and reduced birth weight is independent of all other factors that influence birth weight including race, parity, maternal size, socioeconomic status, and sex of child; it is also independent of gestational age.

3. There is a dose-response relationship between maternal smoking and reduced birth weight; the more the woman smokes during pregnancy, the greater the reduction in birth weight.

4. If a woman gives up smoking early during pregnancy, her risk of delivering a low-birth-weight baby approaches that of a nonsmoker.

5. The ratio of placental weight to birth weight increases with increasing levels of maternal smoking, reflecting a considerable decrease in mean birth weight and a slight increase in mean placental mass; this may represent an adaptation to relative fetal hypoxia.

6. The pattern of fetal growth retardation that occurs with maternal smoking is a decrease in all dimensions including body length, chest circumference, and head circumference.

7. Maternal smoking during pregnancy may adversely affect the child's long-term growth, intellectual development, and behavioral characteristics.

8. Maternal smoking during pregnancy exerts a direct growth-retarding effect on the fetus; this effect does not appear to be mediated by reduced maternal appetite, eating or weight gain.

9. The risk of spontaneous abortion, fetal death, and neonatal death increases directly with increasing levels of maternal smoking during pregnancy; interaction of maternal smoking with other factors which increase perinatal mortality may result in an even greater risk.

10. Excess deaths of smokers' infants are found mainly in the coded cause categories of "unknown" and "anoxia" for fetal deaths, and the categories of "prematurity alone" and "respiratory difficulty" for neonatal deaths; this suggests that the excess deaths are due to problems of the pregnancy, rather than to abnormalities of the fetus or neonate.

11. Increasing levels of maternal smoking result in a highly significant increase in the risk of abruptio placentae, placenta previa, bleeding early or late in pregnancy, premature and prolonged rupture of membranes, and preterm delivery—all of which carry high risks of perinatal loss.

12. Although there is little effect of maternal smoking on mean gestation, the proportion of fetal deaths and live births that occur before term increases directly with maternal smoking level. Up to 14 percent of all preterm deliveries in the United States may be attributable to maternal smoking.

13. The incidence of preeclampsia is decreased among women who smoke during pregnancy; however, if preeclampsia develops in a smoking woman, the risk of perinatal mortality is markedly increased compared to preeclamptic nonsmokers.

14. An infant's risk of developing the "sudden infant death syndrome" is increased by maternal smoking during pregnancy.

15. There are insufficient data to support a judgement on whether maternal and/or paternal cigarette smoking increases the risk of congenital malformations.

16. Infants and children born to smoking mothers may experience more long-term morbidity than those born to nonsmoking mothers; however, studies usually cannot distinguish between the effects of smoking during pregnancy and the effects of the infant's or child's passive exposure to cigarette smoke after birth.

17. Studies in women and men suggest that cigarette smoking may impair fertility.

18. Experimental studies on tobacco smoke, nicotine, carbon monoxide, polynuclear aromatic hydrocarbons, and other constituents of smoke help define pathways by which maternal smoking during pregnancy may exert its aforementioned effects.

Peptic Ulcer Disease

The 1979 Surgeon General's Report included evidence that cigarette smoking in males was significantly associated with the incidence of peptic ulcer disease and increased the risk of dying from peptic ulcer disease by approximately two-fold. The effect of smoking on pancreatic secretion and pyloric reflux demonstrated among men may provide a mechanism by which peptic ulcers develop.

1. Female smokers show a prevalence of peptic ulcer higher than that of nonsmokers by approximately two-fold.
2. The effect of cessation on healing is not known.

Interactions of Smoking with Drugs, Food Constituents and Responses to Diagnostic Tests

Most published studies investigating the effects of cigarette smoking on drug use have been performed on mixed populations; factors specific for women have not been demonstrated to date. It has, however, been clearly demonstrated that women are prescribed and consume more prescription drugs than men.

1. Studies of selected drugs indicate that smoking may affect clinical responses and alter the dose required for an effective therapeutic result.
2. Smoking interacts with oral contraceptive use to increase the risk of myocardial infarction and subarachnoid hemorrhage.
3. Common clinical laboratory parameters are altered in smokers compared to nonsmokers; the health significance of these changes is unknown.
4. Insufficient information exists for assessment of the impact of smoking on the nutritional needs of women.

Psychosocial and Behavioral Aspects of Smoking in Women

1. The percentage of 17–18 year old women who smoke has shown a steady rise between 1968 and 1979. It now appears, however, that the increase in smoking prevalence among all 12–18 year old females has leveled off and begun to decline. Young women born after 1962 show a substantially reduced

initiation of smoking and will probably have a much lower prevalence of smoking as adults.

2. Those young women who do begin to smoke are starting to smoke regularly at a younger age, with more than half of the male and female adolescents who begin to smoke starting before the 10th grade.

3. The earlier tobacco is used and the greater the number of cigarettes smoked per day, the less likely an attempt to quit will be successful.

4. The percentage of women smokers who smoke more than one pack per day is increasing.

5. Adolescent and adult women are more likely to use low-tar and-nicotine cigarettes, smoke fewer cigarettes per day and inhale less deeply than do men, but the difference between the sexes in these patterns of smoking is decreasing. Adolescent and adult black women are more likely to be smokers than their white peers, but they smoke fewer cigarettes per day.

6. Adolescents from low income families, single parent families, and families with lower parental educational levels are more likely to become smokers.

7. Female and male adolescents are more likely to begin smoking if a parent or older sibling also smokes.

8. Adolescent smokers associate with peers who smoke and nonsmokers associate with nonsmoking peers.

9. Adolescent girls overestimate the percentage of their peers who smoke and they have a very positive image of the people in cigarette advertisements, but they are less likely than adolescent boys to see smoking as a social asset.

10. Adolescent girls who smoke tend to be more outgoing but feel less able to influence their future.

11. Adolescents experience stress due to feelings of unattractiveness, incompetency in school achievement and personal relations, limited opportunity for personal growth and concern over future social and economic roles. This stress may be the common mechanism producing the increased rates of smoking in some groups.

12. The factors associated with successful quitting by adolescents of either sex are lower number of cigarettes smoked per day, higher educational aspirations and achievement, greater acceptance of the health risk of smoking, and having more nonsmokers among their friends.

13. It is possible that women and men modify their smoking in order to maintain a constant nicotine level.

14. Women are more likely than men to smoke in order to reduce stress.

15. Women at higher education and income levels are more

likely to succeed in quitting. Additional factors associated with successful quitting are a strong commitment to change, the use of behavioral techniques and reliable social support for quitting. Women have been reported to show lower rates than men of successful cessation following organized cessation programs, a difference which is less apparent in those programs that include social support.

PART I:

PATTERNS OF CIGARETTE SMOKING.

PATTERNS OF CIGARETTE SMOKING

Introduction

This chapter traces the evolution of cigarette smoking among successive generations of American women and men during the twentieth century. The available evidence demonstrates that women have differed from men in their historical onset of widespread cigarette use, in the rate of diffusion of smoking among each new birth cohort, in their intensity of cigarette smoking, and in their use of various types of cigarettes.

Four main conclusions emerge from this analysis. First, although men rapidly took up smoking during the early decades of this century, the proportion of adult female cigarette smokers did not exceed one-quarter until the onset of World War II. The peak intensity of smoking occurred among women born after 1920. Second, as a result of higher past rates of quitting and lower past rates of initiation among men, as well as changes in the type of cigarette consumed, the smoking characteristics of women and men are now becoming increasingly similar. Third, the prevalence of cigarette smoking among adult American women and men is declining. This conclusion applies to all age groups, but with less certainty to the youngest generation of women. Fourth, increasing public awareness of the health consequences of smoking has resulted in significant changes in the nature of the cigarette product. Yet little is known about the effects of these product changes on the initiation, maintenance and cessation of smoking, particularly among women.

Since the last review of cigarette smoking in the 1979 Report of the Surgeon General (24), two new national surveys have been performed under the sponsorship of the National Center for Health Statistics and the National Institute of Education. This chapter relies in part on the recent, preliminary results of these surveys.

The Rise of Cigarette Smoking: 1900–1950

Although the use of cigarettes in the United States was observed as early as 1854 (42,48), consumption did not increase dramatically until after 1900. As shown in Figure 1, per capita consumption of all types of cigarettes increased by more than tenfold from 1900 to 1920. Despite a transient decline during the Great Depression, consumption increased from 665 cigarettes per capita in 1920 to 3,522 cigarettes per capita in 1950 (50).

A continuous, nationally representative series of smoking prevalence rates during the period 1900 to 1950 is not publicly available. Nevertheless, numerous sources can be pieced together to characterize the differential growth of cigarette smoking among women and men.

Figure 2 depicts estimates of the percentage of male and female current cigarette smokers in the greater Milwaukee area, as compiled by the Milwaukee Journal (38). In 1923, the first reported year of this survey, 51.8 percent of males aged 18 years and over smoked cigarettes. Sixty percent of male cigarette smokers also smoked pipes or cigars. In total, 87 percent of adult males used some type of tobacco (38).

Although earlier survey estimates of male smoking rates are unavailable, it appears that the rise of cigarette consumption prior to 1923 reflected both the conversion of established male non-cigarette tobacco users to cigarette smoking and the recruitment of a new generation of younger male smokers during World War I. Innovations in cigarette production and marketing have been cited as influential factors in this rapid growth (39,48,67). Camel cigarettes, a blend of lighter Burley smoking tobaccos with previously dominant Turkish cigarette tobaccos, were introduced in 1913 and within months attained a national market. Two similar brands, Lucky Strike and Chesterfield, followed in 1916 and 1919, respectively (39,48,67). During World War I, the War Industries Board estimated that soldiers of the Allied Armies consumed 60 to 70 percent more tobacco than they had used in civilian life (28,29).

Cigarettes continued to dominate other forms of tobacco among male smokers throughout the 1920s and 1930s. By 1935, 62.5 percent of adult males in the greater Milwaukee area smoked cigarettes (Figure 2), while the percentages of pipe and cigar users had declined substantially. Average cigarette consumption frequency among men smokers increased from 3.7 packs per week in 1923 to 4.8 packs per week in 1935 (38).

Consumption among men accelerated during World War II (Figures 1 and 2). In 1944, more than 25 percent of cigarettes produced in the U.S. were distributed to overseas forces (29), typically for free or at low cost (39), to the point where subsequent shortages developed in the domestic market. By 1948, 67.1 percent of adult males in the Milwaukee area smoked cigarettes (Figure 2). This estimate of the prevalence of cigarette use among urban men is confirmed by other local consumer surveys performed in that year. For example, in 1948, adult male smoking rates were 69.1 percent in Omaha, 67.4 percent in Birmingham, 69.4 percent in Philadelphia, 63.9 percent in Seattle, and 63.4 percent in San Jose (37).

The growth of cigarette smoking among women occurred much later in the face of strong social taboos. Gottsegen noted that "the ultra smart set and women social leaders began to smoke at the turn of the century" (13). By 1906, American "girl stenographers" were reported smoking cigarettes clandestinely (5). By 1919, some younger women in New York were reported smoking at dinner parties "with a trace of defiance" (48). By 1922, New York women were smoking openly on the streets and in bus tops (48).

The first advertisement showing a woman smoking was Lorillard's 1919 publicity for Helmar cigarettes (43,48). In 1926, a young women in a Liggett and Myers' Chesterfieid advertisement did not smoke but pleaded, "Blow some my way" (6). In April, 1927, a Philip Morris advertisement for Marlboro cigarettes noted that "women, when they smoke at all, quickly develop discriminating taste," and that Marlboro cigarettes were as "mild as May" (2). In 1928, a Lucky Strike advertisement urged women to "reach for a Lucky instead of a sweet" (31,39,48). In 1934, Eleanor Roosevelt smoked cigarettes publicly (26). By 1940, handbags and cosmetic compacts were typically designed to hold cigarettes (13).

Although the Milwaukee Journal (38) reported that 16.7 percent of adult women smoked cigarettes in 1934 (Figure 2), prior estimates of women's smoking prevalence are sporadic. Wessel estimated that women consumed 5 percent of all cigarettes in 1924 (66). Moody's Investors Service estimated that women smoked 12 percent of all cigarettes smoked in 1929 (44). The average daily consumption of women smokers, as compared to men smokers, is not documented for that period. If men smokers consumed approximately twice as many cigarettes per day as women smokers (cf. the Milwaukee Journal's 1934 survey report that women's consumption frequency was 135 packs per year as compared to 244 packs per year for male smokers), and if the estimates of male smoking prevalence rates in Figure 2 are taken as nationally representative, and if there were approximately 5 percent more adult males than adult females during the 1920 to 1930 decade (51), then Wessel's estimate yields a 6 percent adult female smoking prevalence in 1924 and Moody's estimate yields a 16 percent prevalence in 1929.

The Milwaukee Journal series in Figure 2 must be interpreted in light of changes in the type of survey respondent and the wording of questions designed to elicit smoking practices (see caption to Figure 2). Moreover, this urban population series may not be representative of all American women. Nevertheless, the publicly available survey data sources are consistent with the conclusion that smoking rates among women did not exceed one-quarter until the onset of World War II.

Based on 10,000 applications for insurance policies during 1930 to 1940, Ley (32) estimated age-standardized smoking rates

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of 63.9 percent of men and 20.8 percent of women aged 15 years and over. In 1935, Fortune Magazine, in the first nation-wide survey (12), reported that 52.5 percent of adult men and 18.1 percent of adult women smoked cigarettes. (See Table 1). Among those under 40 years of age, 65.5 percent of men and 26.2 percent of women were smokers. Among those over 40 years, 39.7 percent of men and 9.3 percent of women were smokers. Urban-rural differences in smoking were significant. The proportion of smokers ranged from 61.4 percent of men and 31.2 percent of women in cities with population over one million, to 44.1 percent of men and 8.6 percent of women in rural areas with population under 2,500. A survey of 250 urban women by the Market Research Corporation in 1937 reported 26 percent regular smokers and an additional 23 percent occasional smokers (47).

After 1940, women's smoking rates accelerated, as new generations of women, particularly younger women in urban areas, entered the labor force (see also title "Occupation and Environment" in this Report). In 1944, the Gallup Poll reported 48 percent adult male smokers and 36 percent adult female smokers (4). In 1949, the Gallup findings were 54 percent male and 33 percent female (4). Local consumer surveys of urban areas in 1948 revealed 37.6 percent adult women cigarette smokers in Milwaukee (see also Figure 2), 34.3 percent in Omaha, 35.6 percent in Birmingham, 46.7 percent in Philadelphia, 38.3 percent in Seattle, and 34.0 percent in San Jose (37). Conover, citing "trade journal" surveys in the three or four years prior to 1950, reported smoking prevalence rates of 65 to 70 percent among men and 40 to 45 percent among women (9).

Although the differential growth of cigarette use among various socioeconomic groups is not well documented, the available data during this period suggest that male smoking rates declined with increasing income, while the relation of women's smoking to income was less clear. The Milwaukee Journal in 1945 noted 58 percent of men with monthly rents over $50 were smokers, and 75 percent of men with rents under $30 per month were smokers (38). Among women, the corresponding proportions were 32 and 37 percent respectively. In Mills and Porter's 1947 survey of Columbus, Ohio (36), 28.3 percent of white females and 64.9 percent white males smoked cigarettes, whereas 36.4 percent black females and 68.9 percent black males smoked cigarettes (estimates calculated from the age distribution data provided in Table 6 of (36)). Kirchoff and Rigdon, in a survey of over 21,000 patients, visitors, and employees of hospitals in Houston and Galveston, noted that 63.2 percent white males, and 33.4 percent white females, 66.3 percent black males, and 32.2 black females smoked cigarettes (30).

All of the above findings reinforce the conclusion that the onset of widespread cigarette use among women lagged behind that of men by 25 to 30 years. This historical delay in the growth of cigarette smoking among women has also been documented for the United Kingdom (8,46,49).

The Emergence of Filtertip Cigarettes: 1951–1963

As shown in Figure 1, total per capita consumption of cigarettes declined during 1953 to 1954. This decline was coincident with the appearance in the popular press of reports seriously suggesting a link between cigarette smoking and lung cancer (10,33,34,40). Thereafter, the consumption of filtertip cigarettes increased rapidly (Figure 1). In 1953 filtertip cigarettes constituted 2.9 percent of cigarette production. By 1958, their share of production had increased to 45.3 percent, and by 1963 it was 58.0 percent (50).

The transient decline during 1953 to 1954 in the number of cigarettes consumed was not clearly matched by a decrease in the proportion of cigarette smokers (27). At least in urban areas, the proportion of women smokers continued to increase. From 1953 to 1958, the prevalence of adult female smoking increased from 42.9 to 45:4 percent in Milwaukee (Figure 2), from 38.4 to 42.6 percent in Omaha, from 47.0 to 50.2 in Washington, D.C., and from 39.6 to 44.4 percent in San Jose (37).

At the same time, both women and men rapidly converted to filtertip cigarettes. By 1958, filter cigarette use prevailed among 61 percent of women smokers and 42 percent of men smokers in Milwaukee, 54 percent of women smokers and 43 percent of men smokers in Omaha, 53 percent of women smokers and 47 percent of men smokers in Washington, D.C., and 59 percent of women smokers and 42 percent of men smokers in San Jose (37). In a nation-wide 1964 survey reported by the National Clearinghouse for Smoking and Health (64), 79 percent of adult female smokers and 54 percent of adult male smokers used filter cigarettes.

Increasing Public Health Awareness: 1964–1979

Per capita consumption reached a peak of 4,336 in 1963 (Figure 1). It declined transiently after the appearance in January 1964 of the first Report of the Advisory Committee to the Surgeon General (52). Per capita consumption continued to decline during the subsequent period of increased publicity concerning the health hazards of smoking (24,27). Since 1975, per capita consumption has declined at an average rate of 1.4 percent an-

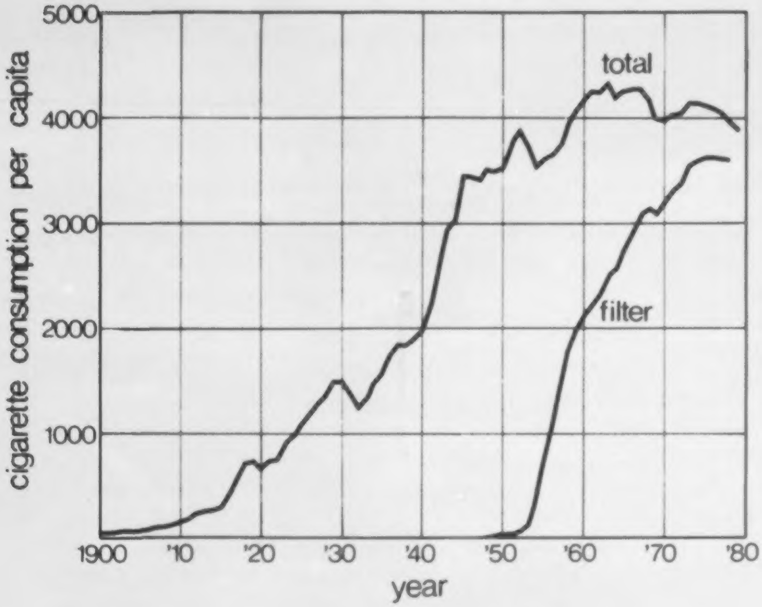

FIGURE 1.—**Annual consumption of cigarettes and filtertip cigarettes per person aged 18 years and over, 1900–1979***

*Total per capita consumption data for 1917–19 and 1940–79 include overseas forces. Total per capita consumption for 1979 is preliminary estimate. Per capita consumption of filtertip cigarettes derived from annual data on the filtertip share of total cigarette production.
SOURCE: U.S. Department of Agriculture (50).

nually. The most recent 1979 estimate of 3,900 cigarettes per capita closely approximates that observed in 1952.

Table 1 summarizes the results of selected, nationally representative surveys of adult cigarette use during the period 1935 to 1979. Except for the Fortune survey of 1935 (12) and the supplement to the Current Population Survey in 1955 (16), these data were collected under the sponsorship of the National Center for Health Statistics. The results of other recent national surveys of adult cigarette use (34,57,58,61,62,64), revealing very similar trends in the prevalence of smoking, were described in the 1979 Surgeon General's Report (24).

Among adult males, the prevalence of regular cigarette use has declined continuously since 1965, with more marked decreases in the intervals 1965 to 1970 and 1976 to 1978. (The absolute standard errors for the National Center for Health Statistics estimates for 1970 to 1976 are less than 0.3 percent. The absolute standard errors for 1978 and 1979 are 0.6 percent.) Among adult women, the direction of change in smoking prevalence is less clear. The estimates for the interval 1976 to 1979, however, suggest a recent downturn. The preliminary 1979 estimate of 32.3 percent for the overall prevalence of adult cigarette smoking among both sexes represents the lowest recorded value in at least 45 years. (The overall prevalence of cigarette smoking in the 1935 Fortune Magazine survey was 37.3 percent among adults of both sexes.)

TABLE 1.—Estimates of the prevalence of regular cigarette smoking among adults, United States, selected national surveys, 1935–1979

Year	Females	Males
1935	18.1	52.5
1955	24.5	52.6
1965	33.3	51.1
1970	31.1	43.5
1974	31.9	42.7
1976	32.0	41.9
1978	29.9	37.0
1979	28.2	36.9

Data for 1978 are revisions of preliminary estimates reported in Harris (26). Data for 1979 are preliminary estimates based on a sample of over 13,000 interviews conducted during January–June 1979, provided by Health Interview Survey, National Center for Health Statistics. 1955 data represent persons 18 years and over. 1976 data represent persons 20 years and over. Estimates for the years 1965, 1970, 1974, 1978 and 1979 represent persons 17 years and over.

SOURCE: Fortune Magazine (12), Haenszel, W. (16), U.S. Department of Health, Education, and Welfare (54–56, 58–59).

These patterns of change in smoking prevalence applied to both white and black adults. For white men, the prevalence of regular smoking declined from 51.5 percent in 1965 to 36.3 percent in 1979. For black men, the prevalence of regular smoking declined from 60.8 percent in 1965 to 42.0 percent in 1979. For white women, smoking prevalence declined from 34.2 percent in 1965 to 28.2 percent in 1979. For black women smoking prevalence declined from 34.4 percent in 1965 to 28.9 percent in 1979. Racial differences in cigarette use are discussed in greater detail in the chapter in this report entitled "Psychosocial and Behavioral Aspects of Smoking in Women."

Although the Milwaukee area data for 1964 to 1979 do not closely match these national estimates, Figure 2 does show a marked decline in smoking rates for both sexes during 1964 to

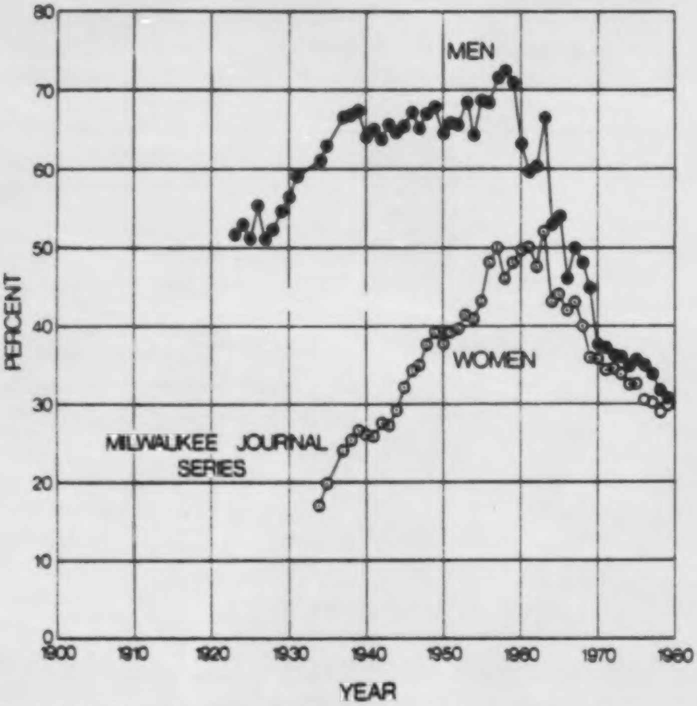

FIGURE 2.—Percentage of adult current cigarette smokers in the greater Milwaukee area, 1924–1979*

*Prior to 1941, the wording of the question eliciting cigarette use and the type of respondent are not recorded. From 1941 to 1954, men were asked, "Do you smoke cigarets?" From 1955 to 1959, all respondents were asked, "Do any men (women) in your household smoke cigarets with (without) a filter tip?" From 1960 to 1965 and in 1967, both men and women were asked "Have you bought, for your own use, cigarets with (without) a filter tip in the past 30 days?" In 1966 and from 1968 to 1979, both men and women were asked, "Have you bought, for your own use, cigarets with (without) a filtertip in the past 7 days?" All percentages reflect adults aged 18 years and over. Data for women from 1976 to 1979 (open circles) represent filtertip cigarette smokers only.

SOURCE: Milwaukee Journal (38).

1970, a deceleration in the decline of smoking prevalence during 1971 to 1975, and a resumption of the decline in prevalence among men in the last four years.

The cessation of cigarette smoking has been a significant factor in explaining this overall decline in smoking prevalence (24). Column (i) of Table 2 presents estimates of the percentage of recent smokers who made a "fairly serious attempt to quit"

TABLE 2.—Estimated rates of attempted and successful quitting among adult, recent cigarette smokers, United States, 1970–1979

	(i) Percent of All Recent Smokers Who Attempted to Quit in Past Year	(ii) Percent of Smokers Attempting to Quit in Past Year Who Reported Successfully Quitting	(iii) Percent of All Recent Smokers Who Reported Successfully Quitting in Past Year
Women			
1970	40.8	21.3	8.7
1975	30.2	19.5	5.9
1978	32.7	18.8	6.2
1979	32.9	21.6	7.0
Men			
1970	44.4	26.4	11.7
1975	28.3	20.1	5.7
1978	29.1	21.5	6.3
1979	31.4	21.3	6.7

1970 and 1975 data from surveys of persons aged 21 years and over, conducted by National Clearinghouse for Smoking and Health. 1978 and 1979 data from the Health Interview Survey of persons aged 17 years and over, conducted by the U.S. National Center for Health Statistics. 1979 data are preliminary estimates based on interviews during January–June of that year.
SOURCE: U.S. Department of Health, Education, and Welfare (54,61,62).

within one year of the interview date. (Recent smokers include all current smokers plus those former smokers reported to have stopped within one year of interview.) Column (ii) shows what proportion of those attempting to quit regarded themselves as former smokers. Column (iii) shows the proportion of all recent smokers (whether or not they attempted or succeeded quitting) who reported themselves as recent former smokers. These data necessarily reflect respondents' self-assessment of both the seriousness of a quit attempt and their degree of success. Nevertheless, they do provide an indication of the representative smoker's annual probability of attempting to quit, the probability of successful cessation given a quit attempt, and the overall annual smoking cessation rate. (The absolute standard errors in Table 4 are approximately 1.0 percent, 1.5 percent, and 0.3–0.5 percent for columns (i), (ii), and (iii), respectively.)

All three indicators of smoking cessation were highest for men in 1970. Although a relatively large proportion of women smokers attempted to quit smoking in 1970 (column (i)), their probability of success in that year was significantly lower than that of men (column (ii)). Quit attempt rates for both sexes (column (i)) declined by 1975, but have increased in 1978 to 1979. With respect to the probability of attempting to quit and the success rate, adult men and women cigarette smokers are now indistinguishable.

Table 3 displays recent changes in the distribution of cigarette brands according to F.T.C. "tar" contents. The proportion of adults smoking cigarettes with F.T.C. "tar" delivery less than 15 milligrams has increased from 9.5 percent of women and 2.9 percent of men in 1970 to 38.5 percent of women and 28.1 percent of me in the first half of 1979. A corresponding increase in the proportion of smokers of cigarettes with F.T.C. nicotine delivery less than 1.0 milligram was also observed.

TABLE 3.—Estimated percentage distribution of adult current regular cigarette smokers according to F.T.C. "tar" content of primary brand, United States 1970–1979

Year	Less Than 5.0 mg	5.0 to 9.9 mg	10.0 to 14.9 mg	15.0 to 19.9 mg	20.0 mg or More
Women					
1970	0.7	2.0	6.8	67.1	23.4
1975	1.2	1.2	15.0	75.1	7.5
1978	5.3	8.8	21.1	59.2	5.7
1979	5.6	9.5	23.4	55.4	6.1
Men					
1970	0.2	0.9	1.8	61.3	28.1
1975	0.6	1.1	11.0	68.1	19.2
1978	3.3	6.2	13.5	63.5	13.6
1979	2.6	8.5	17.0	60.1	11.8

1979 data are preliminary estimates provided by the National Center for Health Statistics. 1970 and 1975 data represent adults aged 21 years and over. 1978 and 1979 data represent adults aged 17 years and over. Estimates exclude those with unknown primary cigarette brand.

SOURCE: U.S. Department of Health, Education, and Welfare (54,61,62).

At the same time, the average daily cigarette consumption of adult smokers has increased. Table 4 shows recent changes in the distribution of reported daily cigarette consumption among current smokers. These data must be interpreted in light of possible underreporting biases (65) and, in particular, a strong tendency for respondents to round off their reported daily consumption to one pack. Nevertheless, the percent of women smoking less than one pack per day has declined, while the proportion smoking more than one pack per day has increased. Except for 1979, a similar trend is observed for men. (The absolute standard errors of the 1978 and 1979 estimates are approximately 1.0 percent.)

The data of Table 4 represent the more recent portion of an apparently long run trend toward increasing daily cigarette consumption among regular smokers. In 1924, Milwaukee men smokers consumed an average of 10 cigarettes per day (38). In 1934, male smokers in Milwaukee consumed an average of 13.4 cigarettes per day, while women smokers consumed 7 per day (38). If cigarette consumption in 1935 was 1,564 per adult (Figure 1 and (50)), and if the overall percentage of adult smokers was 37.3 percent (12), then mean consumption per adult smoker was 11.5 cigarettes per day. If consumption per adult was 3,597 in 1955 and if the prevalence of regular smoking was 37.6 percent (16), then mean consumption per adult in that year was 26.2 cigarettes. The corresponding calculation based on 1979 per capita consumption data and adult prevalence data (Figure 1 and Table 1) yields 33.3 cigarettes per day.

Numerous epidemiological studies and other surveys performed during the period 1950 to 1965 have shown that for both

TABLE 4.—Estimated percentage distribution of adult current cigarette smokers according to reported daily consumption frequency, United States, 1965–1979

Year	Percent Smoking Less Than 15 Cigarettes per Day	Percent Smoking 25 Cigarettes or More per Day
Women		
1965	44.5	13.7
1970	39.1	18.0
1974	38.7	18.5
1976	36.5	19.6
1978	36.0	21.0
1979	34.6	22.4
Men		
1965	29.6	24.5
1970	27.8	27.7
1974	26.3	30.6
1976	24.2	31.1
1978	23.4	34.2
1979	26.4	32.2

Data for 1976 represent persons aged 20 years and over. All other years represent persons aged 17 years and over. Data for 1979 are preliminary estimates based on interviews conducted during January–June of that year, provided by the Health Interview Survey, National Center for Health Statistics.

SOURCE: Harris, J. E. (26), U.S. Department of Health, Education, and Welfare (54–56,58–59).

sexes, especially for women, the proportion of heavy smokers was larger among the younger age groups (14,16,19,20,22, 30,36,61,64). These findings applied to current daily cigarette consumption and lifetime maximum cigarette consumption. They are consistent with the hypothesis that regular smokers in past decades consumed fewer cigarettes per day than contemporary smokers.

The empirical relationships between rates of smoking cessation (Table 2), changes in F.T.C. "tar" and nicotine delivery of cigarettes (Table 3), and increases in daily cigarette consumption (Table 4) are poorly understood (25). It is not known whether smokers of the lowest "tar" cigarettes are more or less likely to attempt to quit, or to succeed in quitting, than smokers of conventional filtertip or nonfilter cigarettes. The extent to which the act of switching to a lower "tar" cigarette may serve as a substitute for quitting may differ among women and men. The observed increase in daily cigarette consumption among current smokers could represent the effect of: higher cessation rates among lighter smokers; an increase in the daily cigarette consumption of continuing smokers; or an increased daily cigarette consumption of new entrants into the smoking population; or a combination of these effects (24). The relationship of these possible mechanisms to the observed increase in the proportion of filtertip cigarette and low "tar" cigarette smokers is not well elucidated.

Exposure to Cigarette Smoke Among Successive Birth Cohorts

Figures 3 and 4 depict estimates of the prevalence of current cigarette smoking from 1900 to 1978 among successive birth cohorts of men and women. Each continuously graphed time series corresponds to individuals born during a particular decade. For example, among women born from 1931 to 1940 (Figure 4), who are now 40 to 49 years old, the prevalence of smoking rose rapidly during the post World War II period and reached a peak of 45 percent by 1963. Thereafter, their overall prevalence of smoking declined to 39 percent in 1978.

These prevalence data were constructed from the reported lifetime smoking histories of over 13,000 respondents to the Health Interview Survey during July to December, 1978. (For related applications of this methodology, see 7,15,27). Although the accuracy of survey recollection of age started smoking, age of smoking cessation, and the duration of significant, temporary periods of abstinence is not known, no particular source of recall bias has been identified (15,16). However, the significantly higher mortality rates of continuing smokers, as compared to

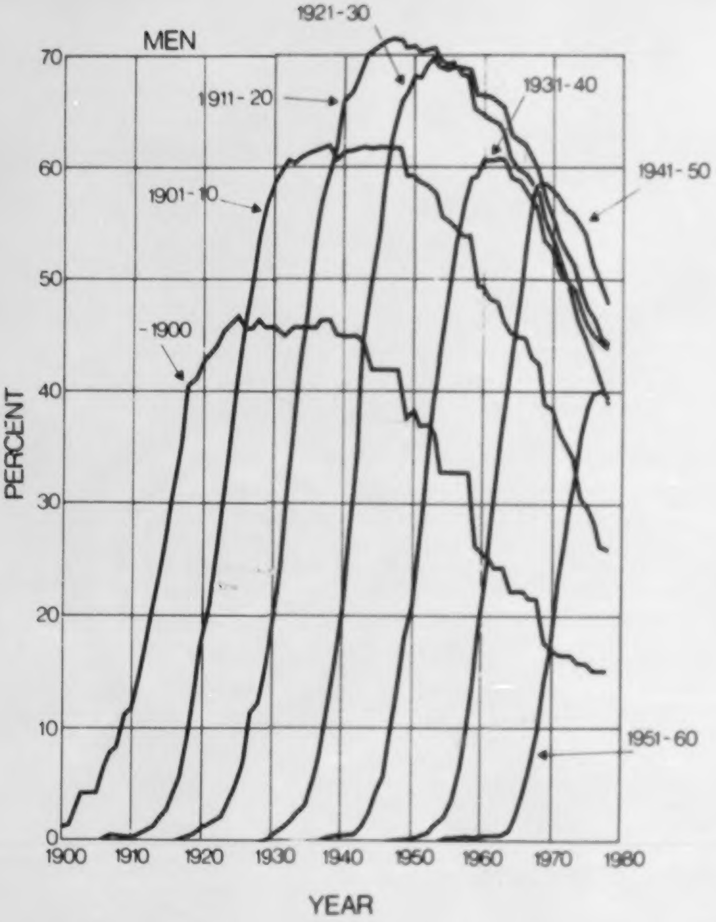

FIGURE 3.—Changes in the prevalence of cigarette smoking among successive birth cohorts of men, 1900–1978

Calculated from the results of over 13,000 interviews conducted during the last two quarters of 1978, provided by Division of Health Interview Statistics, U.S. National Center for Health Statistics.

SOURCE: U.S. Department of Health, Education, and Welfare (60).

nonsmokers or former smokers (1,11,17,18,41,45,46,52), introduces a selection bias that may understate the prevalence of past smoking for the oldest cohorts. For example, on the basis of the insurance life tables recently reported by Cowell and Hirst (11), a male cigarette smoker at age 32 has an estimated 25 percent probability of surviving to age 80, as compared to 49

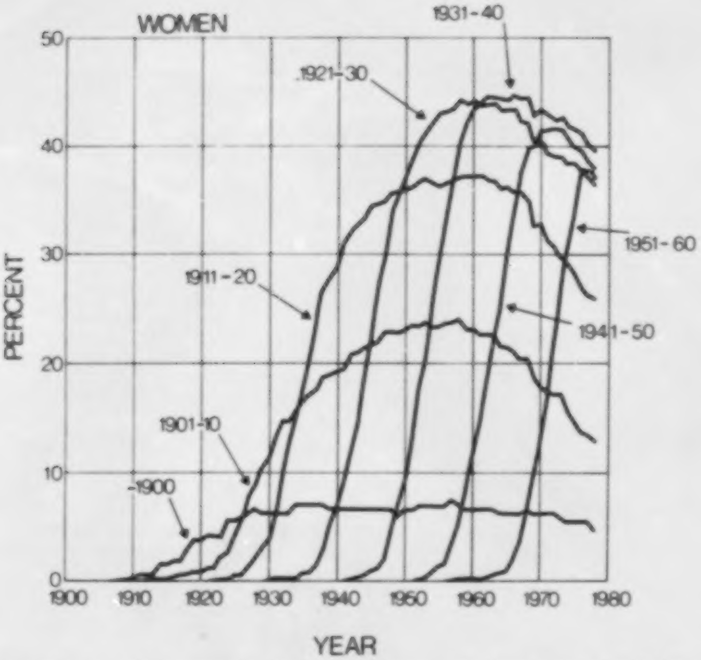

FIGURE 4.—Changes in the prevalence of cigarette smoking among successive birth cohorts of women, 1900–1978

Calculated from the results of over 13,000 interviews conducted during the last two quarters of 1978, provided by Division of Health Interview Statistics, U.S. National Center for Health Statistics.
SOURCE: U.S. Department of Health, Education, and Welfare (60).

percent for a nonsmoker. The estimated probabilities of surviving to age 60 are 80 percent for smokers and 93 percent for nonsmokers, respectively. Therefore, the peak smoking prevalence rate of men born before 1900, calculated from 1978 survey responses to be 46 percent in 1937, could actually have been as high as 65 percent. Since individuals who quit smoking have a higher survival than continuing smokers (18,45), the actual point in time at which smoking rates peaked in this cohort may have been later than 1937. This effect is less likely to be important among men born after 1910, who are now approaching 70 years old. A similar calculation for men born, for example, between 1911 and 1920 reveals that their peak smoking rate may have been understated by at most 2 or 3 percentage points.

This source of bias is likely to be less important for older women. On the basis of age-specific mortality data reported by

Hammond in 1966 (18, Appendix Table 2b), women continuing to smoke cigarettes from age 35 would have an estimated 48 percent chance of surviving to age 80 years, as compared to 54 percent for nonsmokers. The estimated probabilities of survival to age 60 would be 91 percent for smokers and 93 percent for nonsmokers. If these survival data are currently applicable to women smokers and nonsmokers, then the estimated peak prevalence rate of smoking among women born before 1910 could be understated by only one to two percentage points.

Despite these possible biases, the predicted percentages of current smokers in Figures 3 and 4 are consistent with past survey and epidemiological data on the smoking habits of different age groups (12,14–16,19–23,30,35,36,55).

Comparison of Figures 3 and 4 reveals the following conclusions. (a) The most marked differences in smoking prevalence among men and women appeared in those individuals born before 1910, who are now over 70 years of age. (b) Women born between 1921 and 1940, who are now approaching 40 to 59 years of age, experienced the highest smoking prevalence rates. These women have not yet reached the age where the absolute excess deaths of smokers over nonsmokers are expected to become substantial (1). (c) Among successive cohorts of men and women, the age of peak smoking prevalence has declined. Among younger cohorts, the peak smoking prevalence rates are declining, although the effect is less marked for women. Men born between 1911 and 1920 reached a peak smoking prevalence of 71 percent during 1946 to 1948, while those born 1941 to 1950 reached a peak smoking prevalence of 58 percent in 1968 to 1969. Women born 1921 to 1930 reached a peak prevalence of 44 percent in 1958 to 1960, while those born in 1941 to 1950 reached a peak smoking prevalence of 41 percent in 1970 to 1973. (d) Among men born 1951 to 1960, the rate of increase of smoking prevalence was slower than in previous cohorts. This slowing of the diffusion of smoking practices was coincident with the increased publicity concerning the health risks of smoking and the relatively high rate of quitting smoking among adult males in the late 1960s. A similar effect is not clearly discernible for young women in this cohort. In both sexes, among individuals who are now approaching ages 20 to 29, the prevalence of smoking has apparently peaked. Smoking rates among men and women in this age group are now nearly indistinguishable.

Figure 5 depicts the mean age of starting regular smoking among successive birth cohorts, calculated from the same data as for Figures 3 and 4. The age of onset of smoking among women declined continuously during this century, to the point where it is nearly indistinguishable from that of men. As a re-

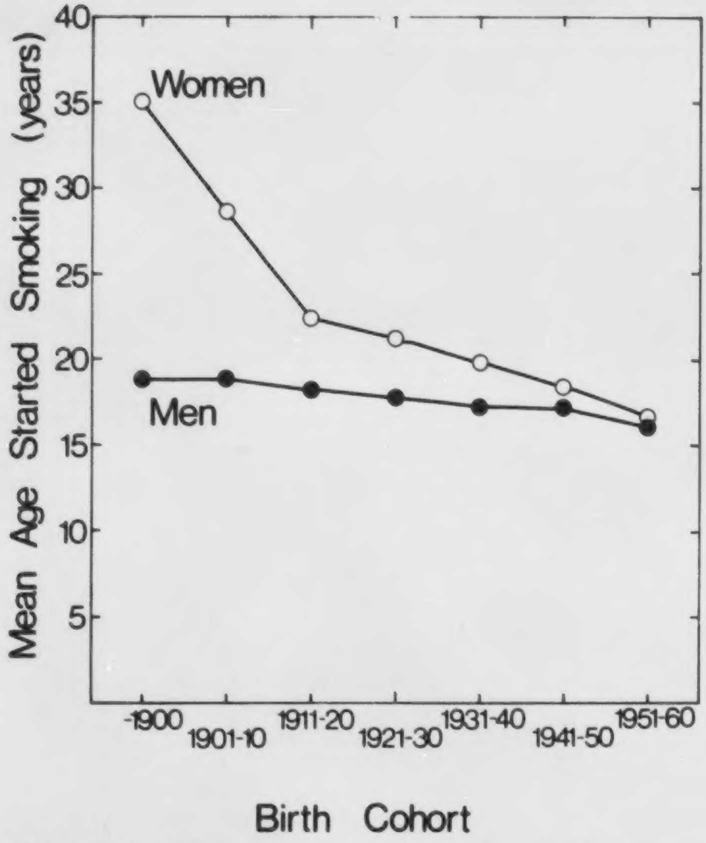

FIGURE 5.—Mean age of onset of regular smoking among successive birth cohorts of women and men
SOURCE: U.S. Department of Health, Education, and Welfare (60).

sult, each successive cohort of lifelong continuing women smokers will have an increasing number of years of exposure to cigarette smoke.

Figure 6 depicts the accumulated years of cigarette smoking per capita, up to 1978, for each birth cohort. These magnitudes correspond to the total areas under each cohort prevalence curve in Figures 3 and 4. Among women, individuals born 1911 to 1920 have thus far experienced the largest total exposure per capita. However, as seen from Figure 4, unless the smoking prevalence rates of women born during 1921 to 1940 decline more rapidly in the future, the lifetime exposure of these latter cohorts is likely to exceed that of the 1911 to 1920 cohort. It is not clear, however, whether the lifetime exposure of men born from 1921 to 1940, now 50 to 69 years of age, will exceed that of previous generations. With each successive cohort, the ratio of female to male exposure increasingly approaches one.

As a result of the rapid diffusion of filtertip cigarettes after 1950 (Figure 1), each successive birth cohort was exposed to a different proportion of filtertip and nonfilter cigarettes. Details of the respondent's past history of cigarette brand use were not obtained in the 1978 Health Interview Survey. Such data, however, are available from a series of over 2,000 interviews of current and former smokers aged 21 years and over, conducted by the National Clearinghouse for Smoking and Health in 1975 (62). Figure 7 depicts, for the same birth cohorts, the proportion of lifetime years of smoking that represents filtertip cigarette use. (The birth dates of the youngest cohorts in Figures 6 and 7 do not match due to differences in survey date and eligible age group.) Among men, there is a distinct, monotonically increasing relation between the proportion of filtertip cigarette exposure and birth date. The corresponding relationship among women born before 1930 reflects their lower smoking cessation rates and, therefore, their continued use of filter cigarettes (62). A woman born in 1925, for example, who began smoking at age 21 (Figure 5), and who switched to filtertip cigarettes in 1957 (Figure 1), has now been smoking filtertip cigarettes for over two thirds of her smoking career and 40 percent of her entire life.

The prevalence of cigarette smoking, age of initiation, lifetime duration of smoking, and the extent of use of various types of cigarettes are not the only measures of cigarette smoke exposure among a particular population. Trends in depth of inhalation, fraction of cigarette actually smoked, and other dimensions of the style of smoking also affect smoke exposure. However, as discussed in the 1979 Surgeon General's Report (24), these are difficult to determine from survey data. In view of the concern over the accuracy of contemporaneous survey reports of daily cigarette consumption (65); past accounts of the time course of daily cigarette consumption would be difficult to assess accurately. Nevertheless, the evidence presented in the previous section is consistent with the conclusion that the average daily cigarette consumption among regular cigarette users has increased among each successive birth cohort.

Cigarette Smoking Among Young Women

The more marked decline in peak smoking prevalence among men born between 1951 and 1960, now approaching 20 to 29 years of age, reflected a slowing in the rate of initiation of smok-

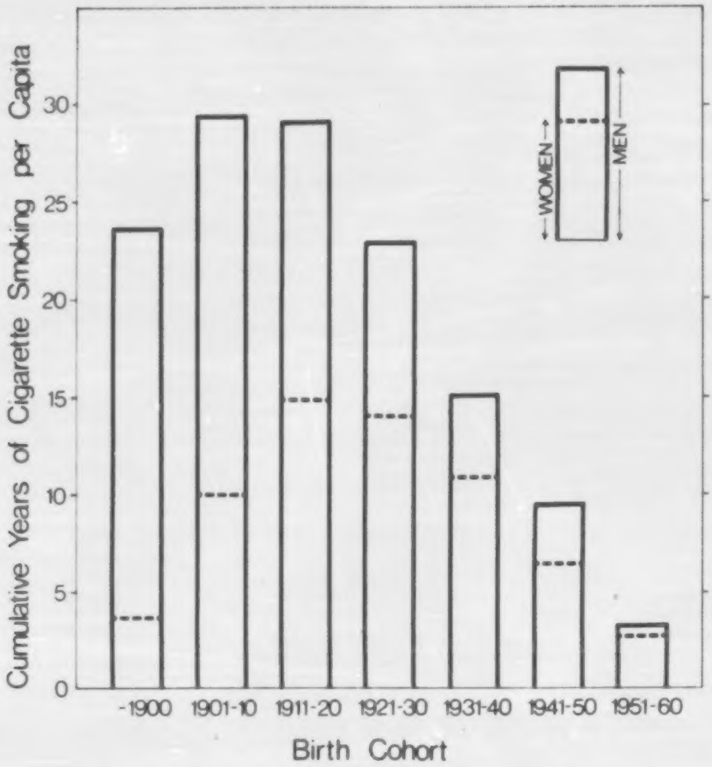

FIGURE 6.—Accumulated years of cigarette smoking per person among successive birth cohorts of women and men, 1978

SOURCE: U.S. Department of Health, Education, and Welfare (60).

ing that was not observed in women of the same age group. This trend appears to be continuing in the next birth cohort.

Table 5 reports the results of nation-wide surveys of teenage cigarette smoking during 1968 to 1979. The most recent survey, conducted by the National Institute of Education during late 1978 and early 1979, presents the preliminary results of over 2,600 telephone interviews of individuals aged 12 to 18 years. In this survey, but not in the others reported in Table 5, women and men 19 years of age were also interviewed. Otherwise, the survey sampling techniques and interview questions regarding smoking practices were the same for all the surveys. (See notes to Table 5).

The data in Table 5 support the conclusion that the rate of initiation of smoking among even the youngest men is declining,

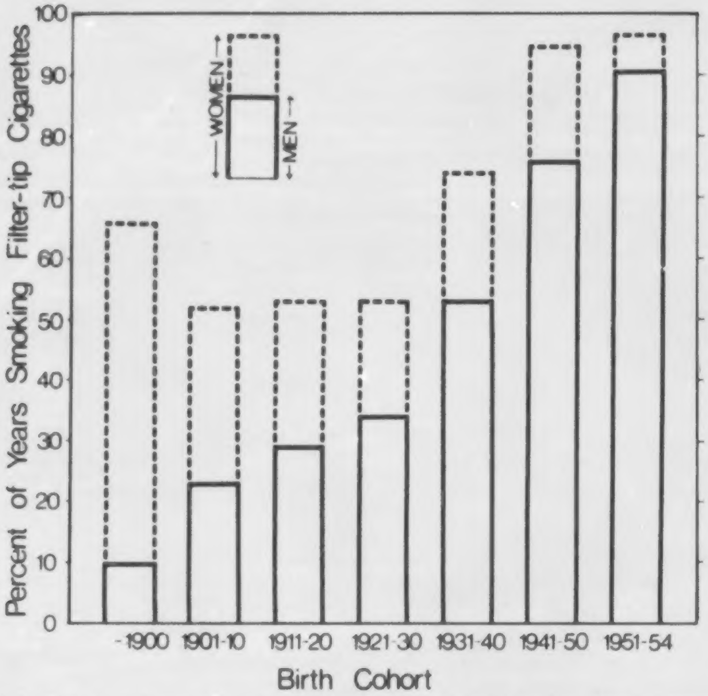

FIGURE 7.—Proportion of years smoking filtertip cigarettes among successive birth cohorts of women and men, 1975

Calculated from the results of over 2,000 smoking histories of men and women who had ever smoked, collected by National Clearinghouse for Smoking and Health.
SOURCE: U.S. Department of Health, Education, and Welfare (62).

an effect that is not present among young women. These results must be interpreted in light of sampling variability. (The absolute standard errors on the 1979 estimates for ages 15–16 and 17–18 are about 2 percent.) As in adult surveys, non-response biases must also be considered. Nevertheless, the findings in Table 5 are consistent with other nation-wide estimates of smoking rates among young women and men. The prevalence of current regular smoking among respondents 17 to 19 years of age in this survey was 28.1 percent for females and 22.8 percent for males. The comparable rates for women and men aged 17 to 19 from the Health Interview Survey were 29.2 percent and 27.5 percent, respectively. An analysis of the growth of smoking prevalence among this group, performed in the same manner as

TABLE 5.—Estimated percentage of current, regular cigarette smokers, ages 12–18, United States, 1968–1979

Year	Ages 12–14	Ages 15–16	Ages 17–18
Females			
1968	0.6	9.6	18.6
1970	3.0	14.4	22.8
1972	2.8	16.3	25.3
1974	4.9	20.2	25.9
1979	4.4	11.8	26.2
Males			
1968	2.9	17.0	30.2
1970	5.7	19.5	37.3
1972	4.6	17.8	30.2
1974	4.2	18.1	31.0
1979	3.2	13.5	19.3

Nation-wide surveys performed by National Clearinghouse for Smoking and Health, 1968–1974, and National Institute of Education, 1979. Current regular smokers in all surveys include all those who smoke cigarettes at least weekly. In 1979, approximately 90 percent of current regular smokers used cigarettes on a daily basis. For 1979 only, 29.7 percent males and 31.9 percent females, aged 19, were reported as regular smokers.

SOURCE: U.S. Department of Health, Education, and Welfare (63).

that of Figures 3 and 4, suggested that smoking rates among this group of women grew rapidly and exceeded those of men by 1975. The future smoking habits of this generation of young women cannot be accurately predicted.

Smoking among adolescent women is discussed in greater detail in the chapter entitled "Psychosocial and Behavioral Aspects of Smoking in Women" in this Report.

Summary

1. Women have differed from men in their historical onset of widespread cigarette use, in the rate of diffusion of smoking among each new birth cohort, in their intensity of cigarette smoking and their use of various types of cigarettes.

2. Men took up cigarette smoking rapidly at the beginning of the twentieth century, especially during World War I. Cigarettes rapidly replaced other forms of tobacco. By 1925, approximately 50 percent of adult males were cigarette smokers. Smoking among men accelerated rapidly during World War II. By 1950, the prevalence of cigarette use among men approached 70 percent in some urban areas.

3. The onset of widespread cigarette use among women lagged behind that of men by 25 to 30 years. The proportion of adult

women smoking cigarettes did not exceed one-quarter until the onset of World War II.

4. Between 1951 and 1963, increasing proportions of women and men smokers converted to filtertip cigarettes. By 1964, 79 percent of adult women smokers and 54 percent of adult men smokers used filter cigarettes.

5. After reaching a peak value of 4,336 in 1963, annual per capita consumption of cigarettes declined in 1964, 1968–70, and in the period since 1975. The most recent estimate of 3,900 cigarettes per capita in 1979 is approximately equal to that observed in 1952.

6. From 1965 to 1978, the proportion of adult men cigarette smokers declined from 51 to 37 percent. The preliminary estimate of adult men's smoking prevalence for 1979 is 36.9 percent. From 1965 to 1976, the proportion of adult women smokers remained virtually unchanged at 32 to 33 percent. Since 1976, the proportion of women smokers has declined to below 30 percent. For 1979, the preliminary estimate of adult women's smoking prevalence is 28.2 percent. The overall smoking prevalence of 32.3 percent for both sexes in 1979 represents the lowest recorded value in at least 45 years.

7. The proportion of adult smokers attempting to quit smoking declined from 1970 to 1975, but increased in 1978–1979. In contrast to past years, the proportions of women and men now attempting to quit smoking, and their reported quitting rates, are indistinguishable. Approximately one in three adult smokers now makes a serious attempt to quit smoking during the course of a year. Approximately one in five of those who attempt to quit subsequently succeed.

8. The proportion of adult smokers using lower "tar" and nicotine brands has increased substantially. In 1979, 39 percent of adult women smokers and 28 percent of adult men smokers reported primary brands with F.T.C. "tar" delivery less than 15.0 milligrams. It is not known whether smokers of the lowest "tar" cigarettes are more or less likely to attempt to quit smoking, or to succeed in quitting, than smokers of conventional filtertip or non-filter cigarettes.

9. The average number of cigarettes smoked by women and men current smokers has increased. The relationship of this finding to recent declines in the average F.T.C. "tar" and nicotine deliveries of cigarettes is not well understood.

10. With each successive generation, the smoking characteristics of women and men have become increasingly similar.

11. Among women, the average age of onset of regular smoking progressively declined with each successive birth cohort—from 35 years of age for those born before 1900, to 16 years of

age among those born 1951 to 1960. The average age of onset of regular smoking among young women is now virtually identical to that of young men.

12. Maximum smoking prevalence rates have declined substantially in recent birth cohorts of men. Men born 1931 to 1940 reached a peak smoking proportion of 61 percent during 1960–62, while men born 1941 to 1950 reached a peak smoking proportion of 58 percent in 1968–69. Men born 1951 to 1960 reached a peak smoking proportion of 40 percent in 1976. Among recent cohorts of women, peak smoking prevalence rates have declined to a much smaller extent. Women born 1931 to 1940 reached a peak smoking proportion of 45 percent in 1966–68, while women born 1941 to 1950 reached a peak smoking proportion of 41 percent in 1970–73. Women born 1951 to 1960 reached a peak smoking proportion of 38 percent in 1976. Among the generation born 1951 to 1960, the proportions of women and men smoking cigarettes are now virtually identical.

13. The proportions of women and men smokers in each age group have declined. Among those born before 1951, this decline in smoking prevalence resulted mainly from smoking cessation. By contrast, the observed decline in smoking prevalence among younger men born 1951 to 1960 has resulted from both smoking cessation and a lower rate of smoking initiation. This decline in the rate of onset of smoking among young men has not been observed for young women.

14. Recent survey data on adolescent smoking habits reveal that by ages 17 to 19, smoking prevalence among women exceeds that of men. This finding supports the conclusion that the rate of initiation of smoking among young men—but not that of young women—is declining. The future cigarette use of the youngest generations of women is uncertain.

15. With each successive birth cohort, the accumulated years of cigarette smoking per woman has progressively approached the accumulated years of cigarette smoking per man. Each successive birth cohort has also experienced progressively smaller sex differences in the fraction of lifetime years of smoking that represents filtertip cigarette use.

16. Among men born during this century, each successive birth cohort has thus far experienced fewer cumulative years of cigarette smoking, higher proportionate exposure to filtertip cigarettes, and lower smoking prevalence rates. This relationship between birth date and cigarette smoke exposure does not hold for women. Women born 1921 to 1940 have experienced substantially higher smoking prevalence rates than earlier generations. Unless they quit smoking in substantial numbers, these women, currently aged 40 to 59, will surpass older women

in total years of cigarette smoking per capita, the total years of nonfilter cigarette smoking per capita, and in the total number of cigarettes smoked. The health consequences of this enhanced exposure to cigarette smoke among women are likely to be more prominent in the coming decades.

References

(1) ADAMS, E.E. Mortality. In: Smoking and Health. A Report of the Surgeon General. U.S. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, January 1979, pp. 2-1 to 2-47.

(2) ADVERTISING & SELLING. Marlboro makes a direct appeal. Advertising and Selling 8:25, March 23, 1927.

(3) AMERICAN INSTITUTE OF PUBLIC OPINION (GALLUP). The Gallup Opinion Index, September 1970, July 1971, July 1972, June 1978.

(4) AMERICAN INSTITUTE OF PUBLIC OPINION (GALLUP). The Gallup Poll Public Opinion, 1935-1971 Series, pp. 477-1501; 1972-1977 Series, pp. 274-1203.

(5) BAIN, J., JR., WERNER, C. Cigarettes in Fact and Fancy. Boston, H.M. Caldwell Co., 1906.

(6) BONNER, L. Why cigarette makers don't advertise to women. Advertising & Selling 7: 21, October 20, 1926.

(7) BURBANK, F. U.S. lung cancer death rates begin to rise proportionately more rapidly for females than for males: a dose-response effect? Journal of Chronic Diseases 25: 473-479, 1972.

(8) CAIRNS, J. The cancer problem. Scientific American 233(5): 64-78, November 1975.

(9) CONOVER, A.G. Discussion of Elmo Jackson's paper. Journal of Farm Economics 32(4, Part 2): 923-924, November 1950.

(10) CONSUMERS UNION. Cigarette smoking and lung cancer. Consumer Reports 19: 54-92, February 1954.

(11) COWELL, M.J., HIRST, B.L. Mortality Differences Between Smokers and Nonsmokers. Worcester, Massachusetts, State Mutual Life Insurance Company of America, October 22, 1979.

(12) FORTUNE MAGAZINE. The Fortune survey. III. Cigarettes. 12(1): 68, 111-116, July 1935.

(13) GOTTSEGEN, J.J. Tobacco. A Study of Its Consumption in the United States. New York, Pitman Publishing Corp., 1940.

(14) GRAHAM, E.A. Primary cancer of the lung with special consideration of its etiology. Bulletin of the New York Academy of Medicine 27(5): 261-276, May 1951.

(15) HAENSZEL, W., SHIMKIN, M.B. Smoking patterns and epidemiology of lung cancer in the United States: are they compatible? Journal of the National Cancer Institute 16(6): 1417-1441, June 1956.

(16) HAENSZEL, W. SHIMKIN, M.B., MILLER, H.P. Tobacco Smoking Patterns in the United States. U.S. Department of Health, Education, and Welfare, Public Health Service, Monograph No. 45, 1956.

(17) HAMMOND, E.C. Life expectancy of American men in relation to their smoking habits. Journal of the National Cancer Institute 43(4): 951-962, October 1969.

(18) HAMMOND, E.C. Smoking in Relation to the Death Rates of One Million Men and Women. National Cancer Institute Monograph 19: 127-204, January 1966.

(19) HAMMOND, E.C., GARFINKEL, L. Changes in cigarette smoking. Journal of the National Cancer Institute 33: 49–64, 1964.

(20) HAMMOND, E.C., GARFINKEL, L. Changes in cigarette smoking 1959–1965. American Journal of Public Health 58(1): 30–45, January 1968.

(21) HAMMOND, E.C., GARFINKEL, L. Influence of Health on Smoking Habits. National Cancer Institute Monograph 19: 269–285. January 1966.

(22) HAMMOND, E.C., GARFINKEL, L. Smoking habits of men and women. Journal of the National Cancer Institute 27: 419–442, 1961.

(23) HAMMOND, E.C., HORN, D. The relationship between human smoking habits and death rates. Journal of the American Medical Association 155: 1316–1328, 1954.

(24) HARRIS, J.E. Cigarette Smoking in the United States,1950–1978. In: Smoking and Health, A Report of the Surgeon General. U.S. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, January 1979, pp. A1–A29.

(25) HARRIS, J.E. Public policy issues in the promotion of less hazardous cigarettes. In: A Safe Cigarette? Cold Spring Harbor Laboratory, Cold Spring Harbor, New York, 1980, pp. 333–340.

(26) HOOVER, I.H. Hail to the chief. Saturday Evening Post, May 5, 1934.

(27) IPPOLITO, R.A., MURPHY, R.D., SANT, D. Staff Report on Consumer Responses to Cigarette Health Information. U.S. Federal Trade Commission, Bureau of Economics, August 1979.

(28) JACKSON, E.L. The Consumption of Tobacco Products: A Descriptive Economic Analysis, United States 1900–1940. Doctoral Dissertation, Harvard University, Cambridge, Massachusetts, 1942.

(29) JACKSON, E.L. Trends in the consumption of tobacco products, United States, 1900–1950. Journal of Farm Economics 32(4, Part 2): 881–893, November 1950.

(30) KIRCHOFF, H., RIGDON, R.H. Smoking habits of 21,612 individuals in Texas. Journal of the National Cancer Institute 16(5): 1287–1304, April 1956.

(31) LEWINE, H. Good-Bye to All That. New York, McGraw-Hill Book Co., 1970.

(32) LEY, H.A., Jr. The incidence of smoking and drinking among 10,000 examinees. Proceedings of the Life Extension Examiners 2: 57–63, 1940.

(33) LIEB, C.W. Can the poisons in cigarettes be avoided? Reader's Digest 63: 45–47, December 1953.

(34) MILLER, L.M., MONAHAN, J. The facts behind the cigarette controversy. Reader's Digest 65: 1–6, July 1954.

(35) MILLS, C.A. Tobacco smoking: some hints of its biological hazards. Ohio Medical Journal 46: 1165–1170, 1950.

(36) MILLS, C.A., PORTER, M.M. Tobacco smoking habits in an American city. Journal of the National Cancer Institute 13: 1283–1297, April 1953.

(37) MILWAUKEE JOURNAL. Consolidated Consumer Analysis. Milwaukee Journal, 1947–1969.

(38) MILWAUKEE JOURNAL. Consumer Analysis of the Greater Milwaukee Market. Milwaukee Journal, 1924–1979.

(39) NICHOLLS, W.H. Price Policies in the Cigarette Industry. Nashville, Tennessee, Vanderbilt University Press, 1951.

(40) NORR, R. Cancer by the carton. Reader's Digest 61: 7–8, December 1952.

(41) PEARL, R. Tobacco smoking and longevity. Science 87(2253): 216–217, March 4, 1938.

(42) PORTER, E.O. The cigarette in the United States. Southwestern Social Science Quarterly 28: 64–75, June 1947.

(43) PRINTERS' INK. Blow some more my way. Printers' Ink 159(2): 20, April 14, 1932.

(44) PRINTERS' INK. Women and cigarettes. Printers' Ink 158(7): 25–27, February 18, 1932.

(45) ROGOT, E. Smoking and mortality among U.S. veterans. Journal of Chronic Diseases 27: 189–203, 1974.

(46) ROYAL COLLEGE OF PHYSICIANS OF LONDON. Smoking or Health. Kent, England, Pitman Medical Publishing Co., Ltd., 1977.

(47) SALES MANAGEMENT. How critical are men of women who smoke and drink? Sales Management 41(6): 36, September 15, 1937.

(48) TENNANT, R.B. The American Cigarette Industry. New Haven, Connecticut, Yale University Press, 1950.

(49) TOBACCO RESEARCH COUNCIL. Statistics of Smoking in the United Kingdom. In: Todd, G.F. (Editor). Research Paper No. 1, 1972, and Supplements 1973–1975.

(50) U.S. DEPARTMENT OF AGRICULTURE, ECONOMIC RESEARCH SERVICE. Tobacco Situation, various issues.

(51) U.S. DEPARTMENT OF COMMERCE, BUREAU OF THE CENSUS. Historical Statistics of the United States, Colonial Times to the Present, 1975.

(52) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE. Smoking and Health, Report of the Advisory Committee to the Surgeon General of the Public Health Service, Public Health Service. Publication No. 1103, 1964, 387 pp.

(53) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL CENTER FOR HEALTH STATISTICS. Changes in cigarette consumption between June 1966 and August 1968. Monthly Vital Statistics Report 19(9, Supplement): December 16, 1970.

(54) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL CENTER FOR HEALTH STATISTICS. Changes in cigarette smoking and current smoking practices among adults: United States, 1978. Advance Data From Vital and Health Statistics No. 52: 1–16, September 19, 1979.

(55) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL CENTER FOR HEALTH STATISTICS. Changes in Cigarette Smoking Habits Between 1955 and 1966. Vital and Health Statistics, Series 10, Number 59, April 1970.

(56) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL CENTER FOR HEALTH STATISTICS. Characteristics of Persons with Hypertension. Vital and Health Statistics, Series 10, Number 121, December 1978.

(57) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL CENTER FOR HEALTH STATISTICS. Cigarette Smoking Status—June 1966, August 1967, and August 1968. Monthly Vital Statistics Report 18(9, Supplement): 1–4, December 5, 1969.

(58) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL CENTER FOR HEALTH STATISTICS. Cigarette Smoking: United States, 1970. Monthly Vital Statistics Report 21(3, Supplement): 1–8, June 2, 1972.

(59) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL CENTER FOR HEALTH STATISTICS. Current Estimates from the Health Interview Survey, United States–1976. Vital and Health Statistics, Series 10, Number 119, November 1977.

(60) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL CENTER FOR HEALTH STATISTICS. (Unpublished data)

(61) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE. NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH. Adult Use of Tobacco, 1970. June 1973.

(62) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH. Adult Use of Tobacco 1975. 1976.

(63) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH. Patterns and Prevalence of Teenage Cigarette Smoking: 1968, 1970, and 1974. July 1974.

(64) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH. Use of Tobacco, Practices, Attitudes, Knowledge, and Beliefs, United States, Fall 1964 and Spring 1966. July 1969.

(65) WARNER, K. E. Possible increases in the underreporting of cigarette consumption. Journal of the American Statistical Association 73(362): 314–318, June 1978.

(66) WESSEL, C.A. The first sixty billions are the hardest for the cigarette industry. Printers' Ink 120(5): 3–6, 137–146, January 31, 1924.

(67) WHITTEN, I.T. Brand Performance in the Cigarette Industry and the Advantage of Early Entry, 1913–74. U.S. Federal Trade Commission, June 1979.

PART II:

BIOMEDICAL ASPECTS OF SMOKING.

MORTALITY

Introduction and Background

Cigarette smoking has been cited as the single most important environmental factor contributing to premature mortality in the United States (17). A great many epidemiological studies support this statement. The emphasis, in general, has been to study males rather than females. Perhaps the main reason for this discrepancy is that, in the past, relatively few women smoked whereas smoking was common among men. The upward trend in lung cancer death rates in males observed in the 1950s by Dorn and others stimulated epidemiologic studies of smoking and health, especially among males (2,3).

According to the 1979 Surgeon General's Report:

> It is important that attention be called specifically to the mortality that females experience as a result of cigarette smoking. There has been an increase in smoking among teenage girls over the past 10 years. At present, the percentages of teenage boys smoking and teenage girls smoking are nearly identical. For some ages, there are more teenage girl smokers than boy smokers. Over the past 10 years, there has been a gradual reduction in the percentage of the adult population that is smoking. Men have quit in greater numbers than women. There has been only a modest drop in the percentage of women who are smoking. In Canada and several European countries, smoking is decreasing among men but increasing among women.

The present report reviews some of the more important prospective epidemiological studies on cigarette smoking and mortality among women.

Mortality Trends

As background, this section reviews mortality levels by sex and color in the United States, by examining recent trends in overall mortality and in three causes of death which have been strongly linked to cigarette smoking—ischemic heart disease, lung cancer and the combined category of bronchitis, emphysema and asthma.*. These trends are displayed in Figures 1 through 4.

For all causes of death (Figure 1), the trend for females was downwards over the entire period from 1950 to 1977 with a somewhat steeper decline in recent years. The trend in death rates among males was essentially flat during most of the 1950s and 1960s, but has been sharply downwards since the late 1960s.

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*The category, chronic obstructive lung disease, may include asthma, a disease which is not causally related to smoking.

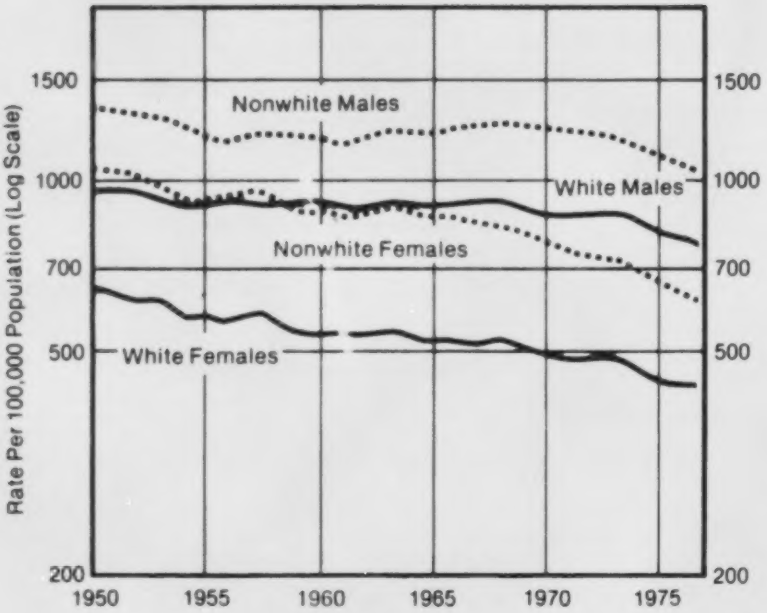

FIGURE 1.—Age-adjusted death rates* for all causes of death by color and sex; United States, 1950–1977

*Adjusted by the direct method to the U.S. population, 1940.
SOURCE: National Center for Health Statistics (9).

For ischemic heart disease, the death rate trend for all sex and color groups was upwards until it flattened in the 1960s. It has been sharply downward since then (Figure 2).

For lung cancer the trend was sharply upwards during the entire period, especially for females (Figure 3).

For bronchitis, emphysema and asthma, the death rate has been sharply upwards for all sex and color groups except nonwhite females. In recent years there appears to be a leveling off for males but not for white females (Figure 4). Other investigators have studied these trends, especially in relation to changes in cigarette smoking habits in the United States and their potential effect upon mortality from the smoking-related diseases (8,12). There are inherent difficulties in interpreting trend data and in particular in relating one trend to another.

Epidemiological Studies

During the past 30 years, there have been eight large pro-

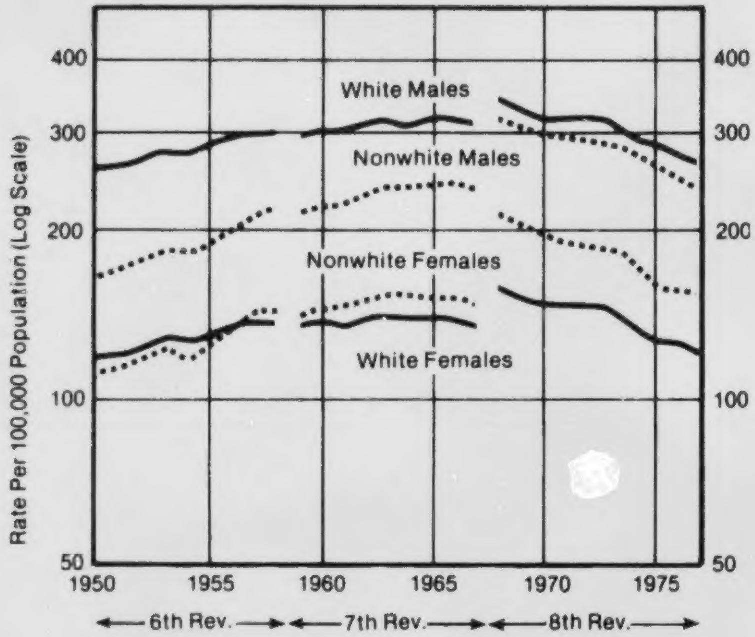

FIGURE 2.—**Age-adjusted death rates* for ischemic heart disease** by color and sex, United States, 1950–1977**

*Adjusted by the direct method to the U.S. population, 1940.
**ICD 6th and 7th Rev. No. 420 and 8th Rev. Nos. 410, 413.
SOURCE: National Center for Health Statistics (9).

spective epidemiological studies specifically designed to delineate the relationship between tobacco smoking and the development of disease. In five of these studies data are available on women as well as men. These studies are outlined below and in Table 1 (1,2,4,5,7,10). To these published results are added unpublished data from two other studies conducted by the National Heart, Lung, and Blood Institute, and from the British Doctors Study.

THE AMERICAN CANCER SOCIETY 25-STATE STUDY (6)

The largest study by far is the American Cancer Society study of men and women in 25 states. In late 1959 and early 1960, the American Cancer Society enrolled 1,078,894 men and women in a prospective study. All segments of the population were

TABLE 1.—Outline of prospective studies of smoking and mortality among women

Authors	Hammond (5)	Cederlof Friberg Hrubec Lorich (1)	Best Josie Walker (4)	Hirayama (7)	Doll Gray Peto (2)	Framingham Heart Study (10)	British-Norwegian Migrant Study British (10)	Norwegian (10)
Type of subjects	Volunteers in 25 states	Probability sample of the Swedish population	Canadian pensioners & dependents	Total pop. of 29 health districts in Japan	British doctors	Sample plus volunteers from Framingham, Mass. (whites)	Probability sample of British & Norwegian migrants to U.S. in 12 states	
Number of female subjects	562,671	27,732	14,226	142,857	6,192	2,873	9,057	5,337
Age range at baseline	35–84	18–69	<30 to 80 +	40 +	25 to 75 +	29–62	45–74	45–74
Year of enrollment	1959	1963	1955	1966	1951	1948	1962	1962
Years of follow-up reported	4	10	6	5	22	26	5	5
Number of female deaths	16,773	1,955	1,794	1,508	1,090	662	588	354
Basic statistical measure	Person-yrs. death rate	Probability of death in 10 yrs.	Probability of death in 6 yrs.	Person-yrs. death rate	Person-yrs. death rate	Probability of death in 26 yrs.	Probability of death in in 5 years	

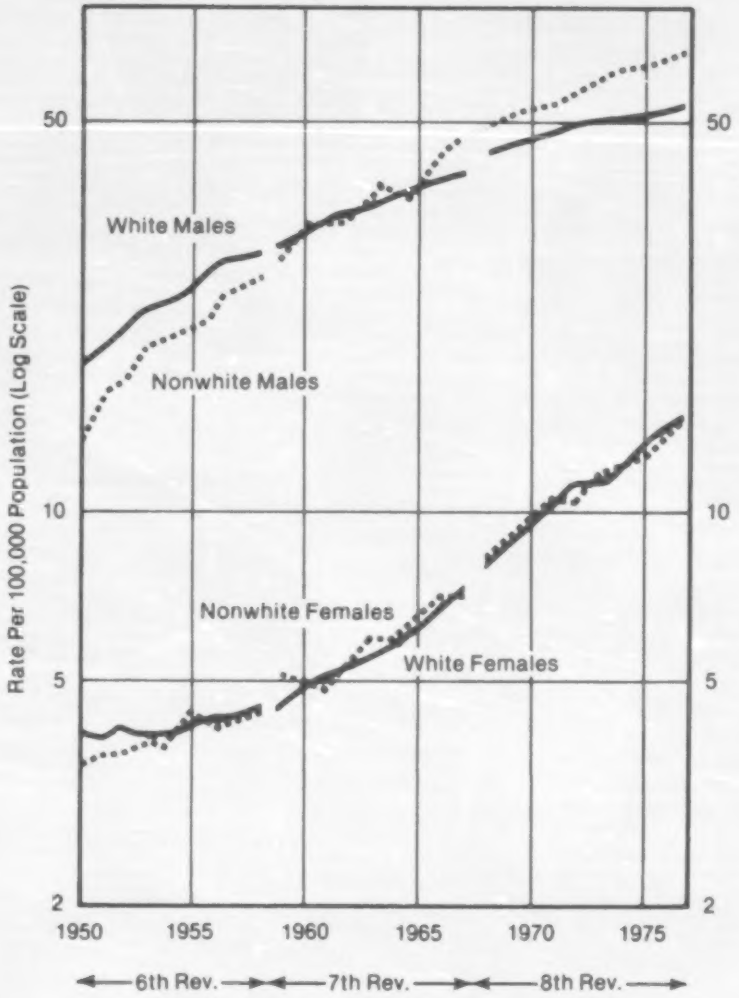

FIGURE 3.—Age-adjusted death rates* for malignant neoplasm of trachea, bronchus, and lung,** by color and sex, United States, 1950–1977

*Adjusted by the direct method to the U.S. population, 1940.
**ICD 6th and 7th Rev. Nos. 162, 163 and 8th Rev. No. 162.
SOURCE: National Center for Health Statistics (9).

included except groups that could not be traced easily. A lengthy initial questionnaire contained information on age, sex, race,

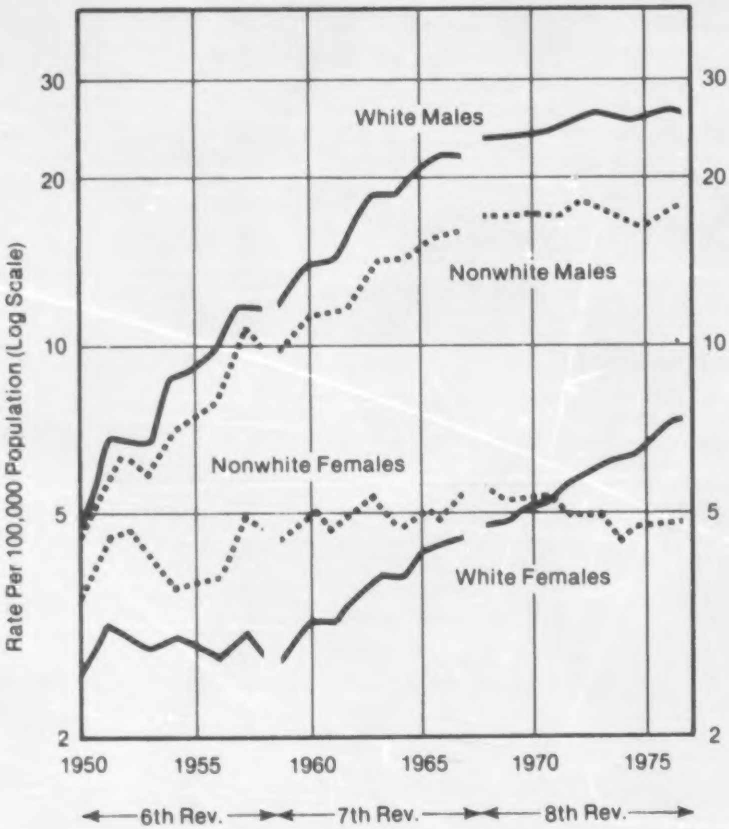

FIGURE 4.—Age-adjusted death rates* for bronchitis, emphysema, and asthma by color and sex, United States, 1950–1977**

*Adjusted by the direct method to the U.S. population, 1940.
**ICD 6th and 7th Rev. Nos. 241,501,502,527.1 and 8th Rev. Nos. 490,493,549.3.
SOURCE: National Center for Health Statistics (9).

education, place of residence, family history, past diseases, present physical complaints, occupational exposures, and various habits. Information on smoking included: type of tobacco used, number of cigarettes smoked per day, degree of inhalation, age at which smoking began, and the brand of cigarettes used from which the "tar" and nicotine content of the cigarette could be calculated. Nearly 93 percent of the survivors were successfully followed for a 12-year period. Only limited data have been published for the 12-year period for women; the main body of published data for women is based on the first 4-year period of the follow-up.

THE SWEDISH STUDY (1)

A national probability sample of 55,000 Swedish men and women was surveyed in 1963, by a mailed questionnaire to which 89 percent of the sample responded. Information was collected on smoking status at the time of the query and at specified intervals during the previous 9 years according to type and amount of smoking and degree of inhalation. The questionnaire identified age, sex, location (urban, nonurban), income, and occuption of each subject. A 10-year follow-up on smoking-related mortality was published in 1975.

THE CANADIAN VETERANS STUDY (4)

Beginning in 1955, the Department of National Health and Welfare, Canada, enrolled 78,000 men (veterans on pension) and 14,000 women (mostly widows of veterans) in a study of smoking-related mortality. Information was obtained on age, detailed smoking history, residence, and occupation. During the 6 years of follow-up, 9,491 of the men and 1,794 of the women died. No recent follow-up has been reported.

JAPANESE STUDY OF 29 HEALTH DISTRICTS (7)

In late 1965, a total of 265,118 men and women in 29 health districts in Japan were enrolled in a prospective study. This represented from 91 to 99 percent of the population aged 40 and older in these districts. This study provides a unique opportunity to examine the relationship of cigarette smoking to death rates in a population with genetic, dietary, and other cultural differences from previously examined Western populations. At the time of the eighth year of follow-up 11,858 deaths had occurred and there were 1,269,382 person-years of observation. For women, however, the main body of published data is based on 5 years of follow-up.

THE BRITISH DOCTORS STUDY (2)

In 1951, the British Medical Association forwarded to all British doctors a questionnaire about their smoking habits. A total of 34,400 men and 6,207 women responded. With few exceptions, all men who replied in 1951 have been followed for 20 years. Further inquiries about changes in tobacco use and some additional demographic characteristics of the men were made in

1957, 1966, and 1972. More than 10,000 deaths have occurred in this population during the past 20 years. For women, published data are available for 11 years of follow-up, and unpublished data are available for 22 years of follow-up.

THE FRAMINGHAM HEART STUDY (10)

The Framingham Study began in 1948 with a cohort of 2,336 white men and 2,873 white women who were age 29 to 62 at the beginning of the study and were residents of Framingham, Massachusetts. Persons were selected by a sample of households plus enlistment of volunteers. These individuals were recalled and examined every 2 years thereafter.

The routine cardiovascular examination consisted of a medical history, physical examination, blood chemistries, body measurements, vital capacity, chest x-ray and a 12-lead electrocardiogram. Mortality and morbidity were documented in detail from the routine biennial examination, hospital records, death certificates, physician records and the next-of-kin.

Information on smoking was obtained at the first examination (and at several thereafter). A series of monographs and over 200 articles on the Framingham Study have now become part of the scientific literature.

Data on the relationship of cigarette smoking to cardiovascular morbidity and mortality, for both men and women, have been reported in the Framingham literature, but the longest reported follow-up period has been 18 years with relatively few deaths having occurred by then, especially among the women (11). Data given below are based on a longer follow-up period, 26 years, and have not been published. The study is presently in its 16th biennial cycle.

THE BRITISH-NORWEGIAN MIGRANT STUDY(10)

In October 1962, morbidity questionnaires requesting information on personal and demographic characteristics, including cigarette smoking, as well as symptoms of cardiorespiratory disease were sent to approximately 32,000 British migrants and 18,000 Norwegian migrants to the United States residing in 12 states. These samples were drawn from the 25 percent random sample of the entire population for which country of birth was recorded in the 1960 United States Census. The 12 states involved contained about three-fourths of the British and Norwegian immigrants to the United States. The response rate to the questionnaire was 86 percent. The respondents were then followed for survivorship and cause of death data for 5 years, from January 1, 1963 through December 31, 1967. The number of morbidity questionnaire respondents and deaths occurring among them from 1963 to 1967 for ages 45 to 74, by sex, were as follows.

	Males		Females	
	Respondents	Deaths	Respondents	Deaths
British	10,103	1,181	9,057	588
Norwegian	5,902	643	5,337	354

Several reports dealing with the prevalence survey and with a related cross-sectional study of mortality, including data on cigarette smoking for women as well as for men, have been published (13,14,15,16). The main results of the prevalence study may be briefly summarized. Four syndromes were considered: "persistent cough and phlegm," "chronic bronchitis," "angina," and "possible infarction." The relation of smoking to the prevalence of these symptoms was clearly demonstrated for women as well as for men. The main results of the cross-sectional mortality study indicated substantial excess mortality for cigarette smokers, as compared to nonsmokers, for both women and men.

Overall Mortality for Females—Cigarette Smokers Versus Nonsmokers

MORTALITY RATIOS

In this report the mortality ratio is the basic means of comparing cigarette smokers with nonsmokers. It is usually obtained by dividing a "death rate" (or other mortality measure) for a classification of smokers by the "death rate" (or other mortality measure) of a comparable group of nonsmokers. The "death rate" may differ markedly from one study to another. In some studies it is calculated by means of person-years and is a 1-year measure; in others it is a probability measure; it may be a 5-year, 10-year or, as in the Framingham Study, a 26-year measure. Differences in mortality ratios may arise because of these factors.

Because of the arithmetic nature of this ratio, there is a tendency for lower ratios to result with higher underlying levels of mortality. For example, with an underlying mortality level of 10 percent per year for nonsmokers, the mortality ratio for a group of smokers can at most be 10 if all the smokers died within the year. With a mortality level of 50 percent for nonsmokers, the maximum possible ratio is 2. Since "death rates" increase with age, there is a tendency for the mortality ratios to decline with age, since its range is restricted.

TABLE 2.—Mortality ratios for female cigarette smokers by number of cigarettes smoked per day and age; females in 24 states

Number of cigarettes per day	Age					Total, 35–84 Age-adjusted[1]
	35–44	45–54	55–64	65–74	75–84	
Nonsmokers	1.00	1.00	1.00	1.00	1.00	1.00
1–9	.90	.95	.99	1.09	1.07	.97
10–19	.97	1.22	1.31	1.18	1.21	1.19
20–39	1.35	1.54	1.46	1.51	.85	1.45
40+	1.56	1.96	1.23	1.42	*	1.63
All Smokers	1.12	1.31	1.27	1.31	1.14	1.26

[1] Adjusted by the direct method using as standard the age distribution of all women.

*Not shown—less than 5 expected deaths.

SOURCE: Hammond, E.C. (5).

TABLE 3.—Mortality ratios for female cigarette smokers by number of cigarettes smoked per day and age; females in the Swedish study

Number of cigarettes per day	Age				Total, 18–69 Age-adjusted
	18–39	40–49	50–59	60–69	
Nonsmokers	1.0	1.0	1.0	1.0	1.0
1–7	1.0	1.6	1.1	.9	1.0
8–15	2.3	2.2	1.7	1.4	1.5
16+	4.5	2.2	1.5	2.2	2.0
All Smokers	1.8	1.9	1.3	1.1	1.2

SOURCE: Cederlof, R. (2).

For simplicity, however, mortality ratios are used throughout this review; it is recognized that these ratios are not strictly comparable from one study to another nor from one age group to another.

AMOUNT SMOKED AND AGE

Overall mortality ratios by amount smoked and age are presented for several of the studies in Tables 2–7. Except for the Swedish study (Table 3), age-adjusted ratios were calculated for each level of smoking in each study. Adjustment was by the direct method, using as standard the age distribution of all

TABLE 4.—Mortality ratios for female cigarette smokers by number of cigarettes smoked per day and age; females in the Canadian study

Number of cigarettes per day	Age				Total, 30+ Age-adjusted[1]
	30–54	55–64	65–74	75+	
Nonsmokers	1.00	1.00	1.00	1.00	1.00
1–9	1.59	1.09	1.05	.92	1.20
10+	2.25	.93	1.20	*	1.43
All Smokers	1.95	1.03	1.10	.95	1.31

[1] Adjusted by the direct method using as standard the age distribution of all women.

*Not shown—less than 5 expected deaths.

SOURCE: Best, E.W.R. (1).

TABLE 5.—Mortality ratios for female cigarette smokers by number of cigarettes smoked per day and age; females in the Framingham Heart Study

Number of cigarettes per day	Age			Total, 29–62 Age-Adjusted[1]
	29–44	45–54	55–62	
Nonsmokers	1.00	1.00	1.00	1.00
<20	1.42	1.21	1.07	1.30
20	1.84	1.48	1.13	1.62
21+	2.25	1.14	*	1.72
All Smokers	1.62	1.28	1.07	1.43

[1] Adjusted by the direct method using as standard the age distribution of all women.

*Not shown—less than 5 expected deaths.

SOURCE: National Heart, Lung, and Blood Institute (10).

women in the particular study. For the Swedish study the age-adjusted values were taken directly from the report.

Mortality ratios shown in Table 2 are considered especially important since they are derived from the study with the largest survivorship experience. Mortality ratios generally rose with the amount smoked for each age group except for the 75 to 84 age group. The age-ratios were .97 for the 1-to-9-cigarettes per day group, 1.19 for the 10-to-19 per day group, 1.45 for the 20–39 group, and 1.63 for the 40-plus group. For all cigarette smokers the age-adjusted mortality ratio was 1.26. By age group, mortality ratios were 1.12 for the 35-to-44 age group,

TABLE 6.—Mortality ratios for female cigarette smokers by number of cigarettes smoked per day and age; British females

Number of cigarettes per day	Age 45–54	55–64	65–74	Total, 45–74 Age-adjusted[1]
Nonsmokers	1.00	1.00	1.00	1.00
< 20	1.49	1.09	.79	1.08
20 +	1.85	1.51	1.55	1.60
All Smokers	1.66	1.25	.98	1.25

[1] Adjusted by the direct method using as standard the age distribution of all women.

SOURCE: National Heart, Lung, and Blood Institute (10).

TABLE 7.—Mortality ratios for female cigarette smokers by number of cigarettes smoked per day and age; Norwegian females

Number of cigarettes per day	Age 45–64	65–74	Total, 45–74 Age-adjusted[1]
Nonsmokers	1.00	1.00	1.00
< 20	1.54	1.07	1.33
20 +	1.41	.89	1.18
All smokers	1.49	1.02	1.28

[1] Adjusted by the direct method using as standard the age distribution of all women.

SOURCE: National Heart, Lung, and Blood Institute (10).

1.31 for the 45–to–54 age group, 1.27 for the 55–to–65 group, 1.31 for the 65–to–74 group and 1.14 for the 75–to–84 age group.

Data from the Swedish study (Table 3) appear to be reasonably consistent with the ACS data in Table 2. The 1–to–7–cigarettes–per–day group had an age-adjusted mortality ratio of 1.0 (compared with .97 for the 1–to–9 group above) and 2.0 for the 16–plus group (compared with 1.63 for the 40–plus group above). For three of the four age groups, the mortality ratios were directly associated with level of smoking. By age group, the highest mortality ratios were observed for the two youngest age groups and the lowest for the two oldest groups. The overall ratio for all cigarette smokers was 1.2.

For the other studies (Tables 4–7) mortality patterns were generally similar in that mortality ratios tended to be highest

TABLE 8.—Mortality ratios for female cigarette smokers by number of cigarettes smoked per day; females in the British Doctors Study

Number of cigarettes per day	Total, Age-adjusted[1]
Nonsmokers	1.00
1-14	0.94
15-24	1.54
25+	1.66
All Smokers	1.23

[1] Based on annual death rates standardized for age.
SOURCE: Cederlof, R. (2).

with heaviest smoking and tended to be lowest at the oldest ages.

For the Japanese study and the British Doctors Study, mortality ratios by amount smoked and age were not reported. However, an overall age-adjusted mortality ratio for female cigarette smokers was reported in the Japanese study, while in the British Doctors Study this ratio was obtained from unpublished data based on 22 years of follow-up (Table 8). We list these along with the overall ratios for the other studies:

Study	Total mortality ratio age-adjusted
American Cancer Society	1.26
Swedish	1.20
Canadian	1.31
Japanese	1.28
British Doctors	1.23
Framingham	1.43
British Migrants	1.25
Norwegian Migrants	1.28

All ratios here are greater than unity. The largest ratio is 1.43 for Framingham. The other seven ratios are close to one another, ranging from 1.2 for the Swedish study to 1.31 for the Canadian study.

DURATION OF SMOKING

Overall mortality ratios for women increased with duration of the smoking habit based on data from the Canadian and

TABLE 9.—Age-adjusted mortality ratios of female cigarette smokers, by number of cigarettes smoked per day and age began smoking; subjects aged 45–54 at start of study. 25-State Study

Number of cigarettes per day	Age began smoking	
	25+	15–24
Nonsmokers	1.00	1.00
1–9	0.95	0.88
10–19	1.17	1.23
20–39	1.33	1.61
40+	**	1.85

**Ratio not shown—less than 10 expected deaths.

SOURCE: Hammond, E.C. (5).

TABLE 10.—Age-adjusted mortality ratios of female cigarette smokers, by number of cigarettes smoked per day and degree of inhalation. Subjects aged 45–54 at start of study. 25-State Study

Number of cigarettes per day	Degree of inhalation of smoke	
	None—Slight	Moderate—Deep
1–9	0.85	1.04
10–19	1.27	1.17
20–39	1.41	1.58
40+	**	2.19

**Ratio not shown—less than 10 expected deaths.

SOURCE: Hammond, E.C. (5).

Swedish studies (1,4). Among Canadian women who smoked for 10 or more years the mortality ratio, adjusted for age, was 1.37 compared to a ratio of 1.08 for women smoking less than 10 years. In the Swedish study an excess risk was found for women smoking 30 or more years (1.4). For those smoking less than 30 years the ratio was 1.0.

AGE BEGAN SMOKING

Table 9 shows mortality ratios for women who were 45 to 54 by number of cigarettes smoked per day and age began smoking (5). Except for the light cigarette smokers (1-to-9-per-day), those taking up the habit at ages 15 to 24 had higher mortality ratios than those who started smoking at older ages.

TABLE 11.—Age-adjusted mortality ratios of female cigarette smokers, by number of cigarettes smoked per day and degree of inhalation and age. 25-State Study

Degree of Inhalation	Age				
	35–44	45–54	55–64	65–74	75–84
Nonsmokers	1.00	1.00	1.00	1.00	1.00
None	**	1.01	1.11	1.12	0.96
Slight	1.22	1.21	1.28	1.26	1.21
Moderate	1.05	1.30	1.32	1.41	**
Deep	1.40	1.78	1.64	**	**

**Ratio not shown—less than 10 expected deaths.
SOURCE: Hammond, E.C. (5).

Mortality data for women smokers, according to age started, are also available from the Swedish study (1); age-adjusted ratios were reported as 1.7, 1.6, and 1.1 for age started less than 17, 17 to 18, and 19 plus, respectively.

INHALATION

Table 10 shows mortality ratios for female cigarette smokers who were 45 to 54 years of age according to number of cigarettes smoked per day and degree of inhalation of smoke (5). No clear pattern emerges. The "moderate-deep" group had higher mortality ratios than the "none-slight" group in two of three comparisons.

Table 11 shows mortality ratios for female cigarette smokers by degree of inhalation and age (5). A fairly consistent general pattern emerges; mortality ratios vary directly with degree of inhalation. This is seen in each age group, except perhaps the 35-to-44 age group.

Mortality data for female cigarette smokers according to inhalation are also available from the Swedish study (1); age-adjusted ratios were reported as 1.1, 1.2, and 1.6 for the no inhalation, light inhalation, and deep inhalation groups, respectively.

"TAR" AND NICOTINE CONTENT OF CIGARETTES

The relationship between overall mortality and the "tar" and nicotine content of cigarette smoke was recently examined by Hammond, et al. (6). In this study, "tar" and nicotine levels (T/N) were defined as follows: "high" T/N, 25.8 to 35.7 mg "tar" and 2.0 to 2.7 mg nicotine; "medium" T/N, 17.6 to 25.7 mg "tar"

TABLE 12.—Adjusted mortality ratios for males and females, by "tar" and nicotine content of cigarettes usually smoked

Sex	Mortality Ratios "High" T/N	 "Medium" T/N	 "Low" T/N
Males	1.00	0.94	0.85
Females	1.00	0.88	0.83
Total	1.00	0.91	0.84

SOURCE: Hammond, E.C. (6).

TABLE 13.—Adjusted mortality ratios for males and females smoking low "tar" and nicotine cigarettes and subjects who never smoked regularly

Sex	Mortality ratios "Low" T/N	 Nonsmokers
Males	1.00	0.61
Females	1.00	0.74
Total	1.00	0.66

SOURCE: Hammond, E.C. (6).

TABLE 14.—Overall mortality ratios of cigarette smokers compared to nonsmokers, by sex and by "tar" and nicotine content of cigarettes usually smoked

Sex	Non-smokers	"Low" T/N	"Medium" T/N	"High" T/N
Males	1.00	1.66	1.85	1.96
Females	1.00	1.37	1.45	1.65
Total	1.00	1.52	1.64	1.80

SOURCE: Hammond, E.C. (6).

and 1.2 to 1.9 mg nicotine; "low" T/N, less than 17.6 mg "tar" and less than 1.2 mg nicotine.

Table 12 shows the overall mortality ratios of male and female smokers by these "tar" and nicotine levels. In this instance, the mortality ratio of the "high" T/N smokers was represented as 1.00 to illustrate the reduction in overall mortality that occurred with lower T/N cigarettes. There was a small reduction in the risk of dying with the use of lower T/N cigarettes. The mortality

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ratio was reduced to 0.91 for the "medium" T/N smokers and was further reduced to 0.84 for the "low" T/N smokers. The mortality ratios were lower for women than for men.

In a separate analysis, a comparison was also made between the mortality ratios of "low" T/N smokers and nonsmokers. These data are presented in Table 13. The mortality ratio of the "low" T/N group was designated as 1.00. Nonsmokers had overall mortality ratios that were considerably less than those of "low" T/N smokers.

The combined data from Tables 12 and 13 are shown in Table 14 where mortality ratios were calculated using nonsmokers as the reference. Combining these data from two separate analyses that are not exactly comparable results in figures that are only approximate.

Hammond also compared death rates of smokers of relatively few (1 to 9) "high" T/N cigarettes with those of smokers who smoked relatively large numbers (20 to 39) of "low" T/N cigarettes (17). The death rates of these two groups were very similar.

Comments

Mortality ratios for women who smoke cigarettes ranged from 1.2 in the Swedish study to 1.43 in the Framingham study. As with men, mortality ratios for women who smoke cigarettes varied directly with amount smoked, depth of inhalation, "tar" and nicotine content of the cigarette and duration of smoking, and varied inversely with the age when smoking was started.

In attempting to study cigarette smoking and mortality among women, a major difficulty is the lack of large-scale epidemiological studies addressed specifically to female populations. The main findings of this review depend heavily on one study, that of the American Cancer Society. For the other studies reviewed here, the numbers of women—and of deaths among them—are often too sparse to permit meaningful statistical analyses. Thus, for example, little can be said about the survivorship experience of women who give up cigarette smoking. We strongly recommend, where possible, extending the length of follow-up of women who are already enrolled in these prospective studies. It is also highly recommended that new studies be conducted that are specifically addressed to women and smoking-related mortality.

Summary

1. The mortality ratio for women who smoke cigarettes is about 1.2 or 1.3.

2. Mortality ratios for women increase with the amount smoked. In the largest prospective study the mortality ratio was 1.63 for the two-pack-a-day smoker as compared to nonsmokers.

3. Mortality ratios are generally proportional to the duration of cigarette smoking; the longer a woman smokes, the greater the excess risk of dying.

4. Mortality ratios tend to be higher for those women who begin smoking at a young age as compared to those who begin smoking later.

5. Mortality ratios are higher for those women who report they inhale smoke than for those who do not inhale.

6. Mortality ratios for women tend to increase with the tar and nicotine content of the cigarette.

7. Mortality ratios for female smokers are somewhat less than for male smokers. This may reflect differences in exposure to cigarette smoke, such as starting smoking later, smoking cigarettes with lower "tar" and nicotine content, and smoking fewer cigarettes per day than men.

8. Women demonstrate the same dose-response relationships with cigarette smoking as men. An increase in mortality occurs with an increase in number of cigarettes smoked per day, an earlier age of beginning cigarette smoking, a longer duration of smoking, inhalation of cigarette smoke, and a higher "tar" and nicotine content of the cigarette. Women who have smoking characteristics similar to men may experience mortality rates similar to men.

References

(1) BEST, E.W.R. A Canadian Study of Smoking and Health. Department of National Health and Welfare, Epidemiology Division, Health Services Branch, Biostatistics Division, Research and Statistics Directorate, 1966, 137 pp.

(2) CEDERLOF, R., FRIBERG, L., HRUBEC, Z., LORICH, U. The Relationship of Smoking and Some Social Covariables to Mortality and Cancer Morbidity. A Ten Year Follow-up in a Probability Sample of 55,000 Swedish Subjects Age 18 to 69. Part I and II. Stockholm, Karolinska Institute, Department of Environmental Hygiene, 1975, 201 pp.

(3) DOLL, R., GRAY, R., PETO, R. Mortality in Relation to Smoking: Observations on Female Doctors. (Unpublished manuscript)

(4) DORN, H.F. The relationship of cancer of the lung and the use of tobacco. The American Statistician 8(5): 7–13, December 1954.

(5) HAMMOND, E.C. Smoking in relation to the death rates of one million men and women. In: Haenszel, W. (Editor). Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases, National Cancer Institute Monograph 19. Department of Health, Education, and Welfare, Public Health Service, National Cancer Institute, January 1966, pp. 127–204.

(6) HAMMOND, E.C., GARFINKEL, L., SEIDMAN, H., LEW, E.A. "Tar" and nicotine content of cigarette smoke in relation to death rates. Environmental Research 12(3): 263–274, December 1976.

(7) HIRAYAMA, T. Smoking in relation to the death rates of 265,118 men and women in Japan. A report of 5 years of follow-up. Presented at the American Cancer Society's 14th Science Writers' Seminar, Clearwater Beach, Florida, March 24–29, 1972, 15 pp.

(8) NATIONAL CENTER FOR HEALTH STATISTICS. Mortality from Diseases Associated with Smoking: United States, 1950–1964. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics, Public Health Service Publication No. 1000-Series 20, 50. 4, October 1966, 45 pp.

(9) NATIONAL CENTER FOR HEALTH STATISTICS, Office of Health Research, Statistics, and Technology, Public Health Service, Department of Health Service, Department of Health, Education, and Welfare. (Unpublished data)

(10) NATIONAL HEART, LUNG, AND BLOOD INSTITUTE. Proceedings of the Conference on the Decline in Coronary Heart Disease Mortality. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, NIH Publication No. 79–1610, May 1979, 399 pp.

(11) NATIONAL HEART, LUNG, AND BLOOD INSTITUTE. Some characteristics related to the incidence of cardiovascular disease and death: Framingham Study, 18-year follow-up. In: The Framingham Study: An Epidemiological Investigation of Cardiovascular Disease. Kannel, W.B., Gordon, T. (Editors). DHEW Publication No. (NIH) 74–599, February 1974.

(12) NATIONAL HEART, LUNG, AND BLOOD INSTITUTE. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, 1979. (Unpublished data)

(13) PEARL, R.B., LEVINE, D.B., GERSON, E.J. Studies of Disease Among Migrants and Native Populations in Great Britain, Norway, and the United States. II. Conduct of Field Work in the United States. National Cancer Institute Monograph 19. Department of Health, Education, and Welfare, U.S. Public Health Service, National Cancer Institute, 1966, pp. 301–320.

(14) REID, D.D. Studies of Diseases Among Migrants and Native Populations in Great Britain, Norway, and the United States. I. Background and Design. National Cancer Institute Monograph 19. Department of Health, Education, and Welfare, Public Health Service, National Cancer Institute, 1966, pp. 287–199.

(15) REID, D.D., CONFIELD, J., MARKUSH, R.E., et al. Studies of Disease among Migrants and Native Population in Great Britain, Norway, and the United States. III. Prevalence of Cardiorespiratory Symptoms Among Migrants and Native Born in United States. National Cancer Institute Monograph 19. Department of Health, Education, and Welfare, Public Health Service, National Cancer Institute, 1966, pp. 321–346.

(16) ROGOT, E. Cardiorespiratory disease mortality among British Norwegian migrants to the United States. American Journal of Epidemiology 108(3): 181–191, 1978.

(17) U.S. PUBLIC HEALTH SERVICE. Smoking and Health. A Report of the Surgeon General. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health. DHEW Publication No. (PHS) 79–50066, 1979, 1251 pp.

MORBIDITY.

MORBIDITY

The relationship between cigarette smoking and morbidity has been summarized in the 1979 Surgeon General's Report. That report contained data from the National Center for Health Statistics Health Interview Survey (HIS) showing the relationship for both men and women between smoking and the prevalence of selected chronic diseases, the incidence of acute illness, days lost from work, days of bed disability, and perceived health status. This section will present additional data from the Health Interview Survey on trends in days lost from work and limitation of activity.

Days Lost from Work

Workers who smoke report losing more work days due to illness and injury than do nonsmokers. This relationship has been observed for both men and women every year that the National Health Interview Survey has included questions on cigarette smoking. For example, in 1965 working women who smoked reported 6.6 work-loss days; working women who had never smoked reported only 4.8 work-loss days (see Table 1). Similarly, in the 1977 HIS women who smoked reported 6.6 days lost from work compared to 5.7 days lost from work by those who never smoked.

The National Clearinghouse for Smoking and Health used the earlier 1965 data to estimate the number of "excess" days lost from work among cigarette smokers. This estimation was obtained by calculating the expected number of work-loss days if all workers had the same work-loss experience as those who had never smoked cigarettes. It was estimated that approximately 20 percent of all work-loss days due to illness and injury could be attributed to the higher rates of loss among current and former smokers (2). The 1979 Surgeon General's Report presented similar calculations, based on 1974 data, and again the estimate was about 20 percent of all work-loss days. These calculations were not sex specific. Certain modifications in the collection procedures have lowered the male response rate for the smoking data and may, thus, make comparisons of more recent data by sex less than ideal. However, the data do show that in 1977 the work-loss rate among women who never smoked was higher than in 1965, while the rates among current smokers remained about the same. This would tend to reduce the number of "excess" days among women attributable to smoking. There has been a slight decrease in work loss among males who never smoked. Former smokers reported fewer work-loss days in 1977 than in 1965. Although the difference in work-loss days between

TABLE 1.—**Days lost from work per year due to illness and injury, per currently employed persons 17 years old and older, by smoking status, sex and age: United States, 1965 and 1977**

	Total[1]	Present Smoker	Former Smoker	Never Smoked
		Percent of work-loss days		
		1965		
Female				
17 +[3]	5.6	6.6	6.7	4.8
17–44	5.5	6.6	6.0	4.5
45–64	6.0	6.7	7.7	5.3
Male				
17 +[3]	5.7	5.9	6.8	4.6
17–44	4.1	4.7	3.6	3.4
45–64	7.8	7.9	9.8	5.6
		1977		
Female				
20 +[3]	6.0	6.6	5.4	5.7
20–44	6.1	6.8	5.4	5.4
45–64	6.4	6.5	5.9[2]	6.5
Male				
20 +[3]	5.3	5.9	6.1	4.2
20–44	5.1	6.0	5.5	4.4
45–64	5.6	5.9	6.2	3.9

[1] Includes unknown smoking status.
[2] Figure does not meet standards of reliability or precision.
[3] Includes ages 65 and over.
SOURCE: National Center for Health Statistics (1).

1965 and 1977 is small, it could be attributed to the assumption that in recent years the former smoker groups have a greater proportion of people who stopped smoking for preventive reasons, that is, before they had experienced serious health consequences.

Further study is needed to determine the association between "excess" days lost from work by smokers and specific diseases. Such an analysis would help explain the economic impact of smoking in the work place.

Limitation of Activity

The Health Interview Survey also regularly collects data on the long-term impact of chronic illness. Respondents were asked if chronic illness limited their activities (3). Estimates of the percent of the population with limitation of activity by cigarette smoking status are shown in Table 2 for 1965 and 1977. Detailed interpretation of trend data is difficult; however, there appears to be a relationship between smoking and the impact of chronic illness. In general, the 1977 data indicate that women under 65 who have ever smoked are more likely to have a limitation of activity than those who never smoked. There are no marked differences between current and former smokers. Among elderly women in 1977, there were no differences in limitations of activity by smoking status.

TABLE 2.—Percent of persons with limitation of activity due to chronic conditions, by cigarette smoking status, sex and age: United States, 1965 and 1977

	Total[1]	Present Smoker	Former Smoker	Never Smoked
	Percent with limitation			
	1965			
Female				
17 +	17.3	12.7	17.3	19.8
17–44	8.3	8.8	9.8	7.7
45–64	19.5	17.4	22.1	20.2
65 +	45.1	39.8	48.6	45.4
Male				
17 +	17.3	15.3	23.0	17.7
17–44	7.3	7.7	8.0	6.2
45–64	20.0	20.9	22.1	15.7
65 +	53.7	52.7	56.3	52.9
	1977			
Female				
20 +	17.6	16.0	18.1	18.3
20–44	8.0	9.2	8.4	7.0
45–64	21.5	24.2	23.9	19.8
65 +	39.2	36.3	35.5	38.8
Male				
20 +	20.0	20.5	24.1	17.6
20–44	9.6	12.4	8.3	7.5
45–64	25.7	27.5	25.7	25.7
65 +	47.5	52.7	47.6	42.5

[1] Includes known smoking status.

SOURCE: National Center for Health Statistics (1).

Cigarette Smoking and Occupation*

The Health Interview Survey provides a considerable data base on cigarette smoking behavior and occupational status.

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*See: "Interaction Between Smoking and Occupational Exposures" in this Report.

The data are available from a national probability sample of about 40,000 households for the years 1965, 1966, 1970, 1974, 1976, 1977, 1978, and 1979. However, only minimal analysis has been conducted on this potentially valuable data base (4). This brief section presents data on smoking patterns for only two of these periods—1970 and 1976. Researchers are encouraged to investigate these data more fully through the purchase of public use data tapes (1). The importance of this data base increases as new evidence becomes available on the increased health risks experienced by smokers in certain occupations. The problems of relatively small sample sizes in high-risk occupations can be partially overcome by combining several years of the HIS data tapes.

Tables 3 and 4 show smoking characteristics of broad occupational groups—i.e., white collar, blue collar, service and farm workers—for 1970 and 1976, respectively. Service and blue collar workers, both women and men, are more likely to smoke than are white collar and farm workers, but the differences are much less among female workers. In 1970, there were virtually no differences among female white collar, blue collar, and service workers; more recently, however, there has been a slight increase in smoking among the latter two groups. Caution should be used in drawing conclusions from these data based on differences of only a few percentage points since such differences can be well within sampling error. White collar workers who smoke tend to be heavier smokers than other types of workers, and this pattern is more marked among female white collar workers.

The proportions of cigarette smokers by more detailed occupational classes are shown in Tables 5 and 6 for 1970 and 1976. Within three of four subgroups of white collar workers—professionals, managers, and sales people—the proportion of smokers among women is the same as for men in the same occupational group. This also appears to be true for laborers, who show the highest levels of smoking among both women and men.

Summary

The 1979 Report of the Surgeon General summarized the information on smoking and morbidity as follows:

1. In general, female current cigarette smokers report more acute and chronic conditions including chronic bronchitis and/or emphysema, chronic sinusitis, peptic ulcer disease, and arteriosclerotic heart disease, than women who never smoked.

2. There is a dose-response relationship between the number

TABLE 3.—Percent distribution of the population 17 years and over by cigarette smoking status, according to sex and occupation category, United States, 1970

Sex and occupation category	Percent distribution							
	Total population[1]	Never smoked	Former smokers	Present smokers	Present smokers—no. of cigarettes per day[2] Total[2]	< 15	15–24	25+
Female								
Total population	100.0	54.0	11.2	34.9	100.0	39.3	42.4	18.2
Total currently employed	100.0	54.3	11.1	34.6	100.0	38.7	43.3	18.0
White collar workers	100.0	53.2	12.6	34.2	100.0	37.6	42.8	19.6
Blue collar workers	100.0	55.1	8.5	36.5	100.0	40.7	44.4	14.9
Service workers	100.0	55.7	9.2	35.2	100.0	41.6	41.0	17.4
Farm workers	100.0	74.3	*7.5	18.6	100.0	*49.2	*33.3	*19.0
Male								
Total population	100.0	28.8	24.9	46.2	100.0	25.8	45.1	29.1
Total currently employed	100.0	28.8	25.2	46.0	100.0	25.5	45.3	29.3
White collar workers	100.0	31.6	29.1	39.3	100.0	23.8	43.4	32.8
Blue collar workers	100.0	24.8	22.4	52.8	100.0	25.5	46.4	28.0
Service workers	100.0	31.1	20.8	48.1	100.0	31.1	43.3	25.6
Farm workers	100.0	40.7	24.8	34.4	100.0	35.5	45.1	19.4

[1] Excludes unknown if ever smoked.

[2] Excludes unknown amount of cigarettes smoked.

*Figure does not meet standards of reliability or precision.

SOURCE: National Center for Health Statistics (1).

TABLE 4—Percent distribution of the population 20 years and over by cigarette smoking status, according to sex and occupation category, United States, 1976

Sex and occupation category	Total population[1]	Never smoked	Former smokers	Present smokers	Present smokers—no. of cigarettes per day[2] Total[2]	<15	15–24	25 +
Female								
Total population	100.0	54.3	13.8	32.0	100.0	36.5	43.8	19.6
Total currently employed	100.0	50.8	13.3	35.9	100.0	36.5	44.0	19.5
White collar workers	100.0	51.1	14.6	34.3	100.0	35.3	42.4	22.3
Blue collar workers	100.0	50.7	10.2	39.0	100.0	38.0	44.3	17.6
Service workers	100.0	49.1	11.9	39.0	100.0	37.9	48.3	13.7
Farm workers	100.0	59.8	*	31.3	100.0	34.6	*	*
Male								
Total population	100.0	29.2	28.9	41.9	100.0	24.2	44.8	31.1
Total currently employed	100.0	29.5	27.1	43.4	100.0	21.9	45.4	32.8
White collar workers	100.0	34.0	29.4	36.6	100.0	20.8	43.6	35.6
Blue collar workers	100.0	24.3	25.3	50.4	100.0	21.2	47.4	31.5
Service workers	100.0	29.4	23.4	47.2	100.0	27.6	40.0	32.4
Farm workers	100.0	34.9	28.2	36.9	100.0	29.4	44.9	25.7

[1] Excludes unknown if ever smoked.

[2] Excludes unknown amount of cigarettes smoked.

*Figure does not meet standards of reliability or precision.

SOURCE: National Center for Health Statistics (1).

TABLE 5.—Estimates of the percentage of current, regular cigarette smokers, adult ages 17 years and over, according to labor force status, occupation, and sex, United States, 1970

	Female			Male		
	Total 17+	17–44	45–64	Total 17+	17–44	45–64
Total	34.9	36.8	33.7	46.2	49.0	44.4
Currently employed	34.6	36.4	33.7	46.0	48.7	44.1
White collar total	34.2	34.9	34.3	39.3	41.1	38.4
Professional, technical and kindred	28.1	29.4	26.3	31.7	32.8	30.6
Managers & administrators except farm	40.8	48.4	38.3	42.8	47.4	40.0
Sales workers	34.6	35.3	35.7	44.9	46.8	46.1
Clerical & kindred workers	35.8	35.9	36.4	43.3	45.2	41.5
Blue collar total	36.5	39.9	33.5	52.8	56.1	49.2
Craftsmen & kindred workers	40.4	44.4	37.0	51.7	56.1	47.2
Operatives and kindred workers	36.5	40.0	33.5	54.7	57.5	50.7
Laborers, except farm	*23.3	*25.6	*20.9	50.9	52.0	52.9
Service	35.2	39.3	33.5	48.1	48.3	51.7
Farm	18.6	*25.9	*15.5	34.4	38.7	37.7
Unemployed	38.4	40.8	32.9	52.3	54.4	53.0
Homemakers	29.7	37.3	32.3	NA	NA	NA

NOTE: Unknown if ever smoked excluded from calculation.
*Figure does not meet standards of reliability or precision.
SOURCE: National Center for Health Statistics (1).

TABLE 6.—Estimates of the percentage of current, regular cigarette smokers, adults ages 20 years and over, according to labor force status, occupation, and sex, United States, 1976

	Female			Male		
	Total 20+	20-44	45-64	Total 20+	20-44	45-64
Total	32.0	36.9	34.8	41.9	47.6	41.3
Currently employed	35.9	37.0	36.1	43.4	46.8	39.7
White collar total	34.3	33.8	36.9	36.6	38.6	35.3
Professional, technical and kindred	29.1	28.6	32.7	30.0	31.1	29.9
Managers & administrators except farm	41.6	42.7	40.8	41.0	46.4	36.1
Sales workers	38.1	37.0	42.6	39.9	42.6	38.0
Clerical & kindred workers	34.8	34.7	36.0	40.4	40.1	44.2
Blue collar total	39.0	43.7	33.6	50.4	54.1	44.3
Craftsmen & kindred workers	40.5	46.9	35.6	48.0	52.1	41.6
Operatives and kindred workers	37.6	42.5	31.2	52.3	55.3	46.2
Laborers, except farm	56.3	52.6	*	53.7	56.9	51.7
Service	39.0	42.8	37.2	47.2	51.1	44.8
Farm	31.3	51.0	*	36.9	45.4	35.0
Unemployed	40.0	41.0	39.2	56.8	59.9	53.8
Usual activity—homemakers	29.0	37.1	32.2	NA	NA	NA

NOTE: Unknown if ever smoked excluded from calculation.
*Figure does not meet standards of reliability or precision.
SOURCE: National Center for Health Statistics (1).

of cigarettes smoked per day and the frequency of reporting for most of the chronic conditions.

3. The age-adjusted incidence of acute conditions (e.g., influenza) for women smokers is 20 percent higher for women who had ever smoked than for nonsmokers.

Additional data from the Health Interview Survey (HIS) is presented:

1. Currently employed women who smoke cigarettes report more days lost from work due to illness and injury than working women who do not smoke.

2. Limitation of activity is reported more commonly among women under the age of 65 who have ever smoked than among those who never smoked.

References

(1) NATIONAL CENTER FOR HEALTH STATISTICS. Standardized Micro-Data Tape Transcript. Department of Health, Education, and Welfare, Public Health Service, DHEW Publication No. 781-213, June 1978.

(2) NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH. Smoking and Illness. Department of Health, Education, and Welfare, Public Health Service, Bureau of Disease Prevention and Environmental Control, National Center for Chronic Disease Control, National Clearinghouse for Smoking and Health, PHS Publication No. 1662, July 1967, 6 pp.

(3) WILDER, C.S. Limitation of activity due to chronic conditions, U.S. 1974. Department of Health, Education, and Welfare, Public Health Service, Health Resource Administration, National Center for Health Statistics, Series 10, No. 111, Public Health Service Pub. No. (HRA) 77-1537, June 1977, 65 pp.

(4) WILSON, R.W. Cigarette smoking, disability days and respiratory conditions. Journal of Occupational Medicine 15(3): 236–240, March 1973.

CARDIOVASCULAR DISEASES

Introduction

While the mortality and morbidity rates of coronary heart disease (acute myocardial infarction and chronic ischemic heart disease) (CHD) are lower for women than men, CHD still represents the major cause of death among women in the U.S. In 1976 the United States recorded 284,055 female deaths as attributable to this cause (Table 2). The difference in mortality rates between the sexes is more marked for acute myocardial infarction, with males of all ages experiencing 189 deaths and females 111 deaths per 100,000 (Table 1). Observed differences by sex in susceptibility to coronary heart disease are not fully understood but appear to be affected by multiple specific risk factors within any demographic group.

McGill and Stern have recently provided an extensive review of sex differences in susceptibility to atherosclerosis in humans and in experimental animals, including an analysis of factors known to predispose to atherosclerosis and its dependent diseases (25).

Mortality Rates

In the United States, the National Center for Health Statistics has reported mortality rates from acute myocardial infarction and chronic ischemic heart disease classified by age, sex, and race, for the years 1968 and 1976 (Tables 1–3) (33). These tables show that mortality rates for acute myocardial infarction among adults up to age 64 are highest for white men and are succeeded by progressively lower rates for other men, other women, and finally, white women. Mortality rates for chronic ischemic heart diseases vary. The rates for white men are second to those for other men and close to those for nonwhite women; again, however, rates for white women are by far the lowest. Both white and nonwhite women show consistently lower rates until extreme old age. However, the differences narrow markedly in age in comparison with those in young adulthood and middle life (Table 1).

Male-to-female mortality ratios for acute myocardial infarction among adults in their 30's and 40's are approximately 5 to 6 for whites and 2 to 3 for nonwhites; among adults in their 70's and 80's, they are roughly 1.6 and 1.4. The actual number of deaths involved is very large; their distribution by age, sex, and race is shown in Table 2. Between 1968 and 1976, a striking decline occurred in the acute myocardial infarction mortality rate for men and women of all ages and races. These are shown

TABLE 1.—Death rates* for acute myocardial infarction and chronic ischemic heart disease for specified age groups, by color and sex; United States, 1968–1976

Year and age	Total			White			All Other		
	Both sexes	Male	Female	Both sexes	Male	Female	Both sexes	Male	Female
1976	Acute myocardial infarction								
All ages	148.8	189.0	110.8	158.7	202.2	117.3	84.0	100.3	69.0
25–34 years	2.8	4.6	1.1	2.6	4.3	0.9	4.2	6.4	2.3
35–44 years	27.0	46.2	8.8	26.6	46.1	7.6	30.4	47.5	10.3
45–54 years	111.7	186.9	41.3	111.8	190.1	37.7	111.2	159.8	68.9
55–64 years	309.5	490.3	147.2	312.2	501.1	142.1	283.2	386.5	194.8
65–74 years	660.1	989.8	406.8	674.5	1,024.7	406.5	524.6	667.9	409.9
75–84 years	1,328.0	1,806.7	1,035.7	1,364.8	1,881.4	1,054.3	917.0	1,061.1	813.0
85 years and over	2,038.0	2,564.7	1,790.3	2,135.0	2,709.6	1,869.9	1,126.5	1,369.1	990.1
1968									
All ages	185.4	243.0	130.6	195.9	258.0	136.7	109.5	133.2	87.7
25–34 years	4.6	7.2	2.2	4.1	6.5	1.7	8.7	13.1	5.0
35–44 years	42.3	70.9	15.2	40.3	69.6	12.1	57.9	81.6	37.9
45–54 years	158.5	267.1	56.8	157.6	270.4	51.3	166.6	236.2	105.3
55–64 years	420.8	668.3	197.1	423.9	684.3	188.4	390.5	512.5	281.0
65–74 years	900.5	1,315.0	574.1	919.8	1,360.8	574.4	706.7	870.1	571.2
75–84 years	1,687.1	2,228.4	1,316.5	1,732.1	2,306.5	1,342.8	1,103.1	1,291.4	961.1
85 years and over	2,911.8	3,570.7	2,553.0	3,012.9	3,715.3	2,637.8	1,782.4	2,163.4	1,526.2

TABLE 1.—Death rates* for acute myocardial infarction and chronic ischemic heart disease for specified age groups, by color and sex; United States, 1968–1976—(Continued)

Year and age	Total			White			All Other		
	Both sexes	Male	Female	Both sexes	Male	Female	Both sexes	Male	Female
1976					Chronic ischemic heart disease				
All ages	150.2	153.5	147.0	155.5	157.7	153.4	115.4	125.4	106.4
25–34 years	1.6	2.4	0.8	1.2	1.9	0.5	4.2	6.1	2.5
35–44 years	12.8	20.3	5.6	10.6	17.5	3.9	27.5	41.0	16.3
45–54 years	57.7	90.9	26.7	50.4	82.6	20.1	116.1	160.7	77.4
55–64 years	173.3	258.5	96.8	159.5	244.3	83.2	302.2	396.1	222.0
65–74 years	487.4	674.8	343.4	467.8	660.5	320.4	672.1	805.8	565.2
75–84 years	1,621.5	1,947.4	1,422.6	1,626.0	1,968.0	1,420.4	1,572.0	1,742.7	1,448.8
85 years and over	4,647.4	4,945.8	4,507.0	4,859.8	5,208.0	4,699.1	2,650.8	2,782.4	2,576.9
1968									
All ages	150.6	156.3	145.1	153.1	158.3	148.2	132.0	141.6	123.3
25–34 years	1.6	2.3	1.1	1.0	1.6	0.4	6.2	7.2	5.3
35–44 years	13.6	20.5	7.1	10.4	17.0	4.0	38.8	49.8	29.5
45–54 years	57.0	85.6	30.2	47.5	76.0	20.7	142.6	175.8	113.3
55–64 years	190.6	273.4	115.7	169.2	253.4	93.0	393.1	468.6	334.8
65–74 years	590.4	769.1	449.7	560.6	742.8	417.9	889.5	1,025.0	777.2
75–84 years	1,826.0	2,075.5	1,655.3	1,833.9	2,093.7	1,657.8	1,724.6	1,858.1	1,628.0
85 years and over	5,523.6	5,636.6	5,468.4	5,695.3	5,831.8	5,629.4	3,605.9	3,736.6	5,518.0

*Rates are deaths per 100,000 population. For acute myocardial infarction, rates are based on deaths assigned to category number 410 of the Eighth Revision of the International Classification of Diseases, adapted for use in the United States, adopted in 1965, and for chronic ischemic heart disease, to category number 412 of this revision

SOURCE: Rosenberg, H.M. (33).

TABLE 2.—Number of deaths* for acute myocardial infarction and chronic ischemic heart disease for specified age groups, by color and sex; United States, 1968 and 1976

Year and age	Total			White			All other		
	Both sexes	Male	Female	Both sexes	Male	Female	Both sexes	Male	Female
1976				Acute myocardial infarction					
All ages	319,477	197,429	122,048	295,613	183,820	111,793	23,864	13,609	10,255
25-34 years	890	718	172	720	598	122	170	120	50
35-44 years	6,223	5,182	1,041	5,338	4,558	780	885	624	261
45-54 years	26,405	21,361	5,044	23,479	19,407	4,072	2,926	1,954	972
55-64 years	62,091	46,516	15,575	56,623	43,072	13,551	5,468	3,444	2,024
65-74 years	93,695	61,038	32,657	86,566	57,004	29,562	7,129	4,034	3,095
75-84 years	89,969	46,395	43,574	84,852	43,912	40,940	5,117	2,483	2,634
85 years and over	40,068	16,132	23,936	37,939	15,201	22,738	2,129	931	1,198
1968									
All ages	369,610	236,017	133,593	342,999	220,517	122,482	26,611	15,500	11,111
25-34 years	1,099	838	261	846	664	182	253	174	79
35-44 years	9,980	8,132	1,848	8,412	7,122	1,290	1,563	1,010	558
45-54 years	36,032	29,368	6,664	32,261	26,860	5,401	3,771	2,508	1,263
55-64 years	76,108	57,387	18,721	69,504	53,287	16,217	6,604	4,100	2,504
65-74 years	109,672	70,564	39,108	101,863	66,205	35,658	7,809	4,359	3,450
75-84 years	100,312	53,838	46,474	95,613	51,436	44,177	4,699	2,402	2,297
85 years and over	36,135	15,711	20,424	34,317	14,824	19,493	1,818	887	931
1976				Chronic ischemic heart disease					
All ages	322,382	160,375	162,007	289,572	143,372	146,200	32,810	17,003	15,807
25-34 years	502	381	121	332	266	66	170	115	55
35-44 years	2,937	2,273	664	2,137	1,734	403	800	539	261

TABLE 2.—Number of deaths* for acute myocardial infarction and chronic ischemic heart disease for specified age groups, by color and sex; United States, 1968 and 1976—(Continued)

Year and age	Total			White			All other		
	Both sexes	Male	Female	Both sexes	Male	Female	Both sexes	Male	Female
45-54 years	13,649	10,391	3,258	10,593	8,426	2,167	3,056	1,965	1,091
55-64 years	34,765	24,525	10,240	28,929	20,996	7,933	5,836	3,529	2,307
65-74 years	69,176	41,612	27,564	60,042	36,745	23,297	9,134	4,867	4,267
75-84 years	109,860	50,010	59,850	101,088	45,932	55,156	8,772	4,078	4,694
85 years and over	91,368	31,109	60,259	86,358	29,217	57,141	5,010	1,892	3,118
1968									
All ages	300,216	151,815	148,401	268,124	135,333	132,791	32,092	16,482	15,610
25-34 years	390	262	128	211	166	45	179	96	83
35-44 years	3,212	2,350	862	2,162	1,734	428	1,050	616	434
45-54 years	12,953	9,412	3,541	9,727	7,545	2,182	3,226	1,867	1,359
55-64 years	34,475	23,481	10,994	27,743	19,732	8,011	6,732	3,749	2,983
65-74 years	71,905	41,270	30,635	62,076	36,135	24,941	9,829	5,135	4,694
75-84 years	108,576	50,145	58,431	101,229	46,689	54,540	7,347	3,456	3,891
85 years and over	68,548	24,801	43,747	64,870	23,269	41,601	3,678	1,532	2,146

*Number of deaths due to acute myocardial infarction are those assigned to category number 410 of the Eighth Revision of the International Classification of Diseases, adapted for use in the United States, adopted in 1965; and for chronic ischemic heart disease to category number 412 of this revision

SOURCE: Rosenberg, H.M. (33).

as percent changes in rate in Table 3. The percent change has been larger at younger ages (Tables 2 and 3). The changes for chronic ischemic heart disease are similar but less dramatic (Table 3).

Atherosclerosis

Differences in heart attack mortality rates among men and women parallel pathology data concerning atherosclerotic plaques of the coronary arteries. The International Atherosclerosis Project systematically collected autopsy observations on persons from 14 geographic locations and 19 ethnic groups in different parts of the world, and found that women from 11 of the 19 groups, when compared to their male counterparts, had as much or even more aortic atherosclerosis. Men over age 39 had more raised plaques in their coronary arteries than women (24).

These findings indicate that the occurrence of coronary plaques was parallel to heart attack rates, but that the occurrence of aortic lesions was not. Coronary plaque severity had a male-to-female ratio of 1.61 among whites and of 1.14 among blacks. Studies of a white population in Sweden (40) and of western Europeans from five locations (18) demonstrate similar findings: a clear excess of coronary atherosclerosis among men and a similar severity of aortic atherosclerosis among men compared to women.

Autopsy studies thus show a selective liability of the male coronary arterial bed for atherosclerosis, as compared to the female, especially among white men but also among men of other races. The pathological findings are congruent with the clinical data on heart attack mortality rates. Autopsy studies also show that, among men or women with manifest coronary heart disease, women patients have roughly the same prevalence of advanced atherosclerotic lesions of the coronaries as men (41). These data suggest that the amount of atherosclerosis necessary to precipitate a heart attack is the same, on the average, in both sexes. This generalization about the amount of coronary atherosclerosis appears to hold for heart attacks at younger and older ages, for recent and old infarcts, and coronary occlusion without infarct, and for stenosis, as well as for complicated and calcified lesions and raised plaques in the coronary arteries (41).

It should be noted that the grading of atherosclerosis at autopsy is not a simple matter because there are several types of lesions and several ways of evaluating or measuring them. Moreover, the development of the different sorts of lesions is

TABLE 3.—Percent change* between 1968 and 1976 in death rates for acute myocardial infarction and chronic ischemic heart diseases for specified age groups, by color and sex: United States

Age	Total			White			All Other		
	Both Sexes	Male	Female	Both Sexes	Male	Female	Both Sexes	Male	Female
				Acute myocardial infarction					
All ages	–19.7	–22.2	–15.2	–19.0	–21.6	–14.2	–23.3	–24.7	–21.3
25–34 years	–39.1	–36.1	–50.0	–36.6	–33.8	–47.1	–51.7	–51.1	–54.0
35–44 years	–36.2	–34.8	–42.1	–34.0	–33.8	–37.2	–47.5	–41.8	–57.0
45–54 years	–29.5	–30.0	–27.3	–29.1	–29.7	–26.5	–33.3	–32.3	–34.6
55–64 years	–26.4	–26.6	–25.3	–26.4	–26.8	–24.6	–27.5	–24.6	–30.7
65–74 years	–26.7	–24.7	–29.1	–26.7	–24.7	–29.2	–25.8	–23.2	–28.2
75–84 years	–21.3	–18.9	–21.3	–21.2	–18.4	–21.5	–16.9	–17.8	–15.4
85 years and over	–30.0	–28.2	–29.9	–29.1	–27.1	–29.1	–36.8	–36.7	–35.1
				Chronic ischemic heart diseases					
All ages	–0.3	–1.8	1.3	1.6	–0.4	3.5	–12.6	–11.4	–13.7
25–34 years		4.3	–27.3	20.0	18.8	25.0	–32.3	–15.3	–52.8
35–44 years	–5.9	–1.0	–21.1	1.9	2.9	–2.5	–29.1	–17.7	–44.7
45–54 years	1.2	6.2	–11.6	6.1	8.7	–2.3	–19.6	–8.6	–31.7
55–64 years	–9.1	–5.4	–16.3	–5.7	–3.6	–10.5	–24.1	–15.5	–33.7
65–74 years	–17.4	–12.3	–23.6	–16.6	–11.1	–23.3	–24.4	–21.4	–27.3
75–84 years	–11.2	–6.2	–14.1	–11.3	–6.0	–14.3	–8.8	–6.2	–11.0
85 years and over	–15.9	–12.3	–17.6	–14.7	–10.7	–16.5	–26.5	–25.5	–26.8

*Percent changes are based on rates per 100,000 population. For 1968 and 1976, rates for acute myocardial infarction are based on deaths assigned to category number 410 of the Eighth Revision of the International Classification of Diseases, adapted for use in the United States, adopted in 1965, and for chronic ischemic heart disease, on category number 412 of this revision

SOURCE: Rosenberg, H.M. (33).

not necessarily parallel. Sternby provides a useful discussion of issues in the grading of atherosclerosis (40). Nevertheless, the major studies noted above provide strong evidence that women have less coronary atherosclerosis on the average than men of the same age in the same population

Risk Factors

Factors present in individuals which correlate with future liability to disease are risk factors for that disease. In the case of heart attack, for example, it has been shown that age, male sex, cigarette smoking, hypertension, elevated blood cholesterol, and several other conditions are positively and independently associated with the probability of heart attack. The level of high-density lipoprotein cholesterol in the serum has a negative correlation with heart attack; that is, higher levels are protective. The various risk factors have been identified for both men and women and have been shown on multivariate analysis to be independent. A combination of risk factors is synergistic, producing an associated risk greater than the simple sum of the individual risks. Although the data for women are much less extensive than for men, they indicate that cigarette smoking is a major risk factor for heart attack in women.

The Effect of Smoking

ATHEROSCLEROSIS

There is little autopsy information about the amount of atherosclerosis in women smokers. Sackett and his associates reported on aortic atherosclerosis among both men and women: of their 450 female subjects, 309 were nonsmokers, 52 smoked less than a half pack per day, and 89 smoked more (34). Mean, age-adjusted aortic atherosclerosis was found to increase in conjunction with the amount and duration of smoking.

A study of the intramyocardial arteries and arterioles of the heart in 13 women and 21 men who were nonsmokers, and 16 women and 27 men who were smokers, indicated that proliferative lesions in intramyocardial arteries were more advanced relative to age in smokers than nonsmokers. It was also found that subendocardial arterioles were thickened in smokers. A separate analysis by sex was not performed, but the authors remarked that the lesions developed as rapidly and as extensively in women as in men in both smoking and nonsmoking groups (28).

Studies of the severity of atherosclerotic plaques in the arteries of women who smoked in comparison with those who did

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TABLE 4.—Coronary heart disease mortality ratios related to smoking—prospective study

Author, year, country	Number and type of populations	Data collection	Follow-up (years)	Number of deaths	Cigarettes/day			Age Variation				
Hammond and Garfinkel, 1969, U.S.A.	358,584 males 445,875 females age 40–70 at entry.	Questionnaire and follow-up of death certificate	6	14,819		M	F	Males				
					NS	1.00	1.00		40–49	50–59	60–69	70–79
					1–9	1.27	0.81					
					10–19	1.00	1.22	NS	1.00	1.00	1.00	1.00
					20–30	1.75	1.52	1.9	1.00	1.50	1.48	1.14
					>40	1.77	0.61	10–19 ...	2.39	2.13	1.82	1.41
								20–30 ...	3.76	2.40	1.91	1.49
								>40	3.51	2.79	1.71	1.47
								Females				
									40–49	50–59	60–69	70–79
								NS	1.00	1.00	1.00	1.00
								1.9	1.31	1.15	1.04	0.74
								10–19 ...	2.04	2.37	1.79	0.98
								20–30 ...	3.62	2.69	2.00	1.27
								>40	+3.31	3.73	+2.02	

Based on 5–9 deaths
NS = nonsmokers, M = males, F = females
SOURCE: U.S. Public Health Service (44,45).

not smoke involve too few subjects to be satisfactory. Investigating the relationship of these arterial lesions and cigarette smoking in women is fundamental to understanding the occurrence of heart attack and other ischemic diseases.

CORONARY HEART DISEASE

Coronary heart disease (acute myocardial infarction and chronic ischemic heart disease) occurs with greater frequency in smoking than in nonsmoking women. The prospective study of Hammond and Garfinkel, published in 1969, included data on approximately 446,000 women between the ages of 40 and 79 (10). The increase in mortality ratios in conjunction with increasing numbers of cigarettes smoked per day for various ages is shown below in Table 4 (43,44). Mortality ratios were higher for younger ages and lower for older ages. The one-pack-a-day smoker's risk of death from heart attack was approximately twice that of the nonsmoker. The prospective data of Shapiro and colleagues are based on a population of 120,000 men and women (36). Using a sampling factor of about one-thirtieth, they examined 4,301 women at risk of a first myocardial infarction between the years 1962 and 1964. The smokers compared with nonsmokers had roughly twice as many rapidly fatal heart attacks and heart attacks that were not fatal within 48 hours. The ratio was approximately 2.9 among younger women aged 45 to 54 and 1.8 for the subjects aged 55 to 64. Heavy smokers had higher ratios, but the data did not permit a detailed study of dose relationships or of the experience of female ex-smokers.

A recent study examined the cause-specific mortality of 6,194 British women physicians over the period 1951 to 1973 (6). Table 5 presents the results of this study in conjunction with the previously published results among male physicians during the same period (7). The clear association of cigarette smoking and ischemic heart disease previously described in males was confirmed in female physicians. For women who reported smoking 15 or more cigarettes per day, mortality due to ischemic heart disease was more than double that of nonsmokers.

Although the results demonstrated a similar effect of smoking in the development of ischemic heart disease in both male and female physicians, the association of smoking with heart disease was less striking in women physicians. Ischemic heart disease was less prominent as a proportional cause of death in this population of women than in male colleagues (16 percent vs. 32 percent of all deaths). Ischemic heart disease mortality was only 26 percent higher for all ever-smoked women than for never-smoked women. However, for females who smoked heav-

TABLE 5.—Death from ischemic heart disease and smoking habits when last asked, British physicians 1951–1973

			Annual Death Rate per 100,000 Persons Standardized for Age					X^2	
					Current Smokers - Dose Per Day				
	Total Popul.	Number of Deaths	Nonsmokers	Ex-smokers	1-14	15-24	> 25	Nonsmokers vs. others	Trend
Women	6194	179	138	126	132	304	292	---	21.14*
						(number of cigarettes)			
Men	34,440	3191	413	533	501	598	677	22.59*	53.56*
						(any tobacco—grams) (1 gram = 1 cigarette)			

*P < 0.001.
SOURCE: Doll, R. (6,7).

ily (≥ 25 cigarettes per day), the relative risk of death from ischemic heart disease was 2.2, a finding consistent with that demonstrated in males, who had a relative risk of 1.6.

In such studies, standardization for amount smoked daily by each of the sexes does not, however, correct for differences in age at initiation of smoking and degree of inhalation. This fact greatly complicates comparison of the magnitude of biologic effect in the two sexes. This "cohort effect" (i.e., unmeasured but documented dissimilarities in total smoking experience) may lead to an erroneous interpretation that cigarette smoking is less damaging to women than to men. This issue cannot be resolved until studies examine the effect of smoking in more recent cohorts of women whose lifetime smoking behavior is more similar to that of men.

Among 26,467 Swedish women observed during a 10-year period, the risk of developing fatal coronary heart disease was significantly higher among smokers than nonsmokers (50). The relative risk was 1.9 at ages 40 to 49 and 1.3 at ages 50 to 59. An extensive mortality study in Japan also reported a highly significant increase in deaths from ischemic heart disease among female smokers, with a mortality ratio for smokers of 1.6 (29).

Coronary heart disease morbidity data are available on women from prospective studies in Framingham, Massachusetts, Tecumseh, Michigan, and the greater New York areas. The Tecumseh data of 1967 do not show a relationship of such morbidity with smoking (Table 6) (8). The Framingham Heart Study found an increased risk for women smokers, but the associations were weak (19,20).

The study of Shapiro and colleagues considered both mortality and morbidity (36). It reported separately on deaths within 48 hours of onset and on all definite myocardial infarctions after that time interval. Using this classification, the incidence of coronary heart disease among women smokers was distinctly higher than it was among nonsmokers.

While there is some variability in the strength of this association, the data from the various prospective studies of mortality and morbidity from coronary heart disease establish smoking as a positive correlate, or risk factor, for women. However, the risk ratios tend to be smaller than for men at a given level of cigarette consumption in all age groups. This trend may result from the different smoking patterns reported by men and women who smoke the same number of cigarettes per day (6,7,25). Men generally begin smoking at an earlier age and have thus smoked for a longer time period than women. Men also inhale more often than women and are more likely to smoke more than half of a cigarette. These smoking styles would ex-

TABLE 6.—Coronary heart disease morbidity as related to smoking

Author, year, country	Number and type of population	Data collection	Follow-up years[1]	Number of incidents[2]	Cigarettes/day[3]			Pipes, cigars	
Epstein, 1967, U.S.A.	6,568 male and female residents of Tecumseh, Mich.	Initial medical examination and repeat follow-up examinations.	4	96 male, 92 female CHD including deaths, angina, and myocardial infarctions	Males			Males	
						40–59	60 and over	40–59	
					NS	1.00 (1)	1.00 (7)	SM	1.80 (2)
					EX	6.33 (10)	1.27 (11)		
					Cigarettes	5.20 (36)	1.90 (23)	60 and over	
								SM	0.80 (6)
					Females				
						40–59	60 and over		
					NS	1.00 (21)	1.00 (47)		
					EX	0.89 (3)	1.31 (5)		
					Cigarettes	1.02 (14)	0.42 (2)		

[1] Reexamination of patients was spread over 1½–6 year period, but data are reported in terms of 4-year incidence rates.

[2] Actual number of CHD incidents derived from data on incidence and total in smoking class.

[3] Risk ratios—actual number of CHD incidents shown in parentheses. SM = smokers, NS = nonsmokers, EX = ex-smokers.

SOURCE: U.S. Public Health Service (45).

pose men to a larger dose of smoke per cigarette and a larger lifetime amount than that experienced by women.

Case control and retrospective studies of women who have had heart attacks have suggested an increased incidence of heart attack among smokers. For example, a case control study of 55 women who had heart attacks before age 50 (an uncommon event in women) found that 89 percent were smokers in contrast to 55 percent in a control group without myocardial infarction. Heavy smokers (35 or more cigarettes per day) had an estimated myocardial infarction rate approximately 20 times that of the nonsmokers. As far as possible, women using oral contraceptives and those with other identifiable risk factors were excluded from the study (37).

Spain and his associates conducted a retrospective autopsy study of women who had died suddenly of coronary heart disease and compared this verified diagnosis to the women's smoking habits as reported by the closest living relative (38). Only witnessed sudden deaths were included in the data. Comparisons were made between women who had died of coronary heart disease and women who died suddenly of causes other than heart attack. It was found that 62 percent of the women suffering sudden cardiac death were heavy smokers in contrast with only 28 percent of the control group. For those who smoked heavily, the mean age at death was 19 years younger than that of nonsmokers; lighter smokers died at an intermediate mean age.

In a retrospective study emphasizing psychosocial variables, Talbott and associates reported on 64 white women who died suddenly of arteriosclerotic heart disease (42). They found that women who died suddenly smoked more cigarettes than the comparison group. The relative risk for those smoking more than a pack a day compared with those smoking less than a pack a day was 3.9 ($p < .004$).

Smoking, as well as other risk factors, raises the already somewhat higher risk of myocardial infarction among women who use oral contraceptives. During the child-bearing years, the use of oral contraceptives doubles the risk of myocardial infarction; women who both smoke and use oral contraceptives have approximately 10 times the risk of women who neither smoke nor use oral contraceptives (14). These issues are considered below in a separate section.

Cessation of Smoking and "Tar" and Nicotine Content of Cigarettes

Existing data are inadequate to determine the effect of smoking cessation on the incidence of coronary heart disease in women. Hammond and associates have reported that mortality rates from coronary heart disease were lower in women who smoked low-"tar" and low-nicotine cigarettes (as sold in the 1960s) than in those who smoked medium level products, and still lower than for those who smoked high-"tar" and high-nicotine products; even so, the mortality rate for those women smoking low-"tar", low-nicotine products was significantly higher than that of nonsmokers (11).

Evidence considered below suggests that stopping smoking is beneficial in the treatment of women suffering from peripheral vascular disease.

ANGINA PECTORIS

The Framingham Heart Study reported that there was a positive association between smoking and angina pectoris among men but not among women (20). In an extensive study conducted in New York City, Shapiro and colleagues reported a positive association between the development of angina pectoris and smoking among men and a nonsignificant positive trend among women (37). Among patients with angina pectoris, smoking lowers the exercise threshold for the onset of angina (46). Only male patients have been studied thus far; equivalent data apparently have not been published for women with angina and angiographically proven coronary atherosclerosis.

CEREBROVASCULAR DISEASE

The incidence of stroke as a manifestation of cerebrovascular disease appears to be somewhat greater in men than in women, but the difference is small (21,30,43).

In an autopsy assessment of cerebrovascular atherosclerosis, Sternby reported more atherosclerosis of the common carotid artery and the carotid sinus in men than women. There was also more intracranial atherosclerosis of certain vessels in men than women. However, using the area-grading method, no sex difference was found in total intracranial atherosclerosis (40). The International Atherosclerosis Project also reported a slight excess of cerebrovascular atherosclerosis among males (24). On the whole, the available pathological evidence suggests a minor increase in cerebrovascular atherosclerosis among men in comparison with women, although some studies fail to confirm this conclusion (see 40).

It is not clear whether smoking is a risk factor among women for the development of atherothrombotic stroke. Kannel has discussed the issue and the current literature in some detail (19). The Framingham Heart Study has reported a dose-related

TABLE 7.—Deaths from cerebrovascular disease related to smoking

Author, year, country	Number and type of population	Data collection	Follow-up years	Number of deaths due underlying to CVD as cause	Mortality ratios				
Hammond and Garfinkel, 1969, U.S.A.	358,584 males 445,875 females 40–79 years of age at entry.	Questionnaire and follow-up of death certificate	6	4,099		Age			
					Cigarettes/day	40–49	50–59	60–69	70–79
						Males			
					Never smoked	1.00	1.00	1.00	1.00
					1–9	2.79	1.95	1.30	0.95
					10–19	1.14	1.48	+1.44	0.92
					20–30	2.21	2.03	1.62	1.22
					>40	1.64	2.40	1.72	+0.68
						Females			
					Never smoked	1.00	1.00	1.00	1.00
					1–9	1.50	1.26	1.26	0.83
					10–19	2.60	2.70	2.15	+0.57
					20–30	2.90	2.67	1.83	1.28
					> 40	+5.70	+3.52	—	—

SOURCE: U.S. Public Health Service (44,45).

correlation between the incidence of atherothrombotic stroke and cigarette smoking in men but not in women. The extensive prospective study of Hammond and Garfinkel, which involved almost 446,000 women and recorded 1,905 deaths from cerebrovascular disease during a six-year period, found that smoking was a positive correlate for such mortality (10); in both men and women, the mortality ratio was increased by roughly 2 or 2.5 times (Table 7) (44,45).

That some of these deaths may have involved subarachnoid hemorrhage rather than brain infarction, is suggested by a recent report that found the incidence of subarachnoid hemorrhage to be positively associated with smoking for both men and women (2). The relative risk for men was 3.9 and for women, 3.7. The association appeared to relate to hemorrhage from ruptured cerebral aneurysms rather than to other conditions that may give rise to subarachnoid hemorrhage. A synergism between smoking and the use of oral contraceptives and subarachnoid hemorrhage is noted below (31). The Japanese study cited in the discussion of ischemic heart disease has also reported on 366 deaths from cerebrovascular disease among women who smoked (29). The risk ratios for subarachnoid hemorrhage and cerebral hemorrhage were both significantly increased among women smokers ($p<.001$) as was the risk rate for the category, "other forms of cerebrovascular disease" ($p<.05$).

ARTERIOSCLEROTIC PERIPHERAL VASCULAR DISEASE

Clinicians have noted that arteriosclerotic peripheral vascular disease is more common in men than women. Sternby has reported from autopsy studies that men generally have somewhat more atherosclerosis of the femoral and pelvic arteries than women (40).

Kannel has reviewed the relationship of smoking to the incidence of arteriosclerotic peripheral vascular disease (19). In the Framingham Heart Study the incidence of peripheral vascular disease was increased among smokers of both sexes; cigarette smoking was as strong an independent risk factor in women as in men. Heavy smokers had a threefold increased incidence.

Weiss studied 245 women with arteriosclerotic peripheral vascular disease (49). Ex-smokers who had not smoked for 5 years or more had nearly a normal risk ratio of 1.06; those who had not smoked for the last 1 to 5 years had a risk of 1.70; continuing smokers of less than a pack a day, 5.15; pack a day smokers, 11.53; and those smoking more than a pack a day, 15.56 (relative to nonsmokers, 1.00). The increased risk was particularly associated with proximal (aortoiliac) disease, and there was less association with distal (femoropopliteal) disease. Age-standardized relative risk ratios for those smoking a pack a day were 30.06 for proximal and combined proximal and distal disease and 6.32 for distal disease alone.

A retrospective study of 217 patients who underwent arterial reconstructive procedures of various kinds for peripheral vascular disease has been reported by Myers and colleagues (27). Diabetics were excluded from the report. There were 164 male and 53 female patients. The late patency rate of the vascular reconstruction was followed for 1 to 4 years. The authors reported that the number of cigarettes smoked before surgery did not influence the outcome, but cessation of smoking after surgery had a favorable impact. There were no significant differences in outcome between men and women. The patency rate 4 years after aortofemoral surgery was 90 percent in those who smoked five or fewer cigarettes per day after surgery and 75 percent in those who smoked a greater amount. Following femoropopliteal reconstruction, the 2-year patency rates were 95 percent for those who stopped smoking, 75 percent for those smoking as many as 15 cigarettes per day, and 65 percent for those who continued to smoke more than 15 cigarettes per day.

AORTIC ANEURYSM

Studies have not been reported for women with respect to atherosclerotic aortic aneurysm and smoking. Deaths for women are about one-fifth those for men (10).

HYPERTENSION

Smoking is not associated with an increased prevalence of essential hypertension in men or women (39). However, smoking does combine with hypertension (and other risk factors) as a risk factor for heart attack, synergistically compounding the risk.

Two recent case control studies of rapidly progressive, severe or malignant hypertension have found that there is an overrepresentation of smokers among patients with this uncommon phase of hypertension (3,13). In one study of 82 patients who developed malignant hypertension, 67 were smokers. Thirty-three of those were women. In the study, 77 percent of the female patients with malignant hypertension smoked, and only about 44 percent of those with essential hypertension and of the general female population smoked. The difference is highly significant. A similar and parallel study of 48 patients with malignant hypertension contained 33 men and 15 women; 25 men (76 percent) and 8 women (53 percent) were smokers compared with 44 percent and 30 percent, respectively, of a group of 44 men and 44 women with nonmalignant hypertension. The difference is significant for men but does not reach significance for women.

VENOUS THROMBOSIS

The section of the 1979 Surgeon General's Report dealing with venous thrombosis noted a case control study by Vessey and Doll of 84 women who had venous thromboembolism (45). There was no significant relationship to smoking, although there was a trend (p =0.08) reasonably attributable to chance (46). Similarly, Lawson, Davidson, and Jick reported no association with smoking among 60 premenopausal women who used oral contraceptives and who had uncomplicated venous thromboembolism (22).

The issue is reopened, however, by a recent paper derived from the Walnut Creek Contraceptive Drug Study. The authors analyzed 38 cases of venous thromboembolic events among the approximately 16,700 women followed in the study. These women were matched with 8,174 controls from the same cohort, providing each case with 61 to 559 comparison subjects. The relative risk of cigarette smoking was 2.6 with a one-sided p value of less than 0.01. On multivariate analysis, the smoking effect was independent and remained significant. Of the 17 idiopathic cases of thromboembolic disease, 65 percent occurred in smokers, while 33 percent of the controls were smokers. The relative risk for smokers was 4.2. Both smoking and oral contraceptive use were independent risk factors for venous thromboembolic disease in this cohort of women (32).

The same section of the 1979 Surgeon General's Report noted a controversy about whether smokers who suffered myocardial infarction had a relative protective effect from leg vein thrombosis in the immediate post infarction period (45). The authors did not provide an analysis for each sex.

A recent investigation of women undergoing gynecologic operations has studied the incidence of deep vein thrombosis of the leg in relation to smoking. In the prospective study of 231 women, their smoking habits during the month before the operation were determined. The occurrence of deep vein thrombosis (DVT) was assessed by the radioactive fibrinogen technique, with routine scans on the first, third, and sixth postoperative days. Of the 231 patients, 99 smoked and 132 did not smoke. Eight of the smokers (8.1 percent) and 29 of the nonsmokers (22 percent) developed DVT. Following an analysis of other factors, the authors concluded that smoking provided an apparent "protective" effect against postoperative DVT, based on the fact that smokers constituted only 21 percent of the patients with DVT. They also noted that the women who developed DVT weighed more than those who did not and that smokers who developed CVT were more overweight than nonsmokers with DVT (5).

In a continuing prospective study of the relationship of blood clotting and blood thrombogenic properties to ischemic heart disease, Meade and associates have reported on a number of blood coagulation variables and their relationship to smoking among 1,426 men and 638 women in England (26). Forty-three percent of the men and 36 percent of the women were smokers. Smoking was not found to have an effect in women on factors V or VII, fibrinogen, fibrinolytic activity, antithrombin III, platelet adhesiveness, or platelet count. Smoking decreased fibrinolytic activity in men and decreased factor VIII activity in both men and women. Oral contraceptive users were found to show an increase in fibrinolytic activity only if the women were nonsmokers.

HIGH-DENSITY LIPOPROTEIN

High-density lipoprotein (HDL) is a protein complex that transports cholesterol in the blood. A higher level of HDL is correlated with a reduced risk of heart attack. It has been observed that women who smoke have lower levels of HDL than expected (1,4,9).

Oral Contraceptive Use, Smoking, and Cardiovascular Disease

The association of oral contraceptive use and an increased incidence of certain cardiovascular disorders has attracted much interest. Smoking has emerged as a strong synergistic risk factor, and an additional study has focused on smoking as an independent risk factor.

The effects of smoking and of estrogen and progestin contraceptives on the level of high-density lipoprotein in women have been studied by Bradley and associates. They measured serum HDL among almost 5,000 women between the ages of 21 and 62 (4). They reported that the use of oral estrogens raised the level of HDL significantly above the level in nonusers while progestin use lowered it. Combination drugs tended to change the HDL level according to their relative estrogen-progestin formulation. The average HDL concentration was reduced by smoking. Among nonsmoking women the HDL concentration was 63.7 ± 16.8 mg/dl. This was reduced by 2.2 mg/dl for those smoking half a pack per day; and by 7.3 mg/dl for those smoking one or more packs per day. A reduction in the HDL level among women who smoked was also reported from Holland. This study found an independent negative association with the HDL level among oral contraceptive users (1).

It has been reported from long-term studies that women using oral contraception have a two to threefold statistically significant increase in risk of venous thromboembolic disease when compared to those using other forms of contraception (47). This study concluded that smoking did not significantly increase the incidence of venous thromboembolism (46). By contrast, the Walnut Creek Study reported that smoking contributed to venous thromboembolism among both users and nonusers of oral contraceptives (32). Conclusions about the effect of smoking on venous thromboembolic phenomena, therefore, must be regarded as uncertain at this time since there are few relevant studies and they provide somewhat contrary conclusions.

In 1973, the Collaborative Group for the Study of Stroke in Young Women estimated that the relative risk of cerebral ischemia or thrombosis was approximately nine times greater for women who use oral contraceptives than for those who do not. A detailed analysis of smoking was not presented, but one of the study's striking findings was the high proportion of women with stroke who currently or at some time smoked cigarettes regularly (73.8 percent), compared with smoking rates of 43.4 percent among neighborhood controls aged 17 to 44. The study also found an increase in hemorrhagic strokes among white women. Almost half of the hemorrhagic strokes were attributable to bleeding from congenital aneurysms leading to subarachnoid hemorrhage (5). Recently an association between smoking and aneurysmal subarachnoid hemorrhage in both men and women has been documented (2).

The Walnut Creek Contraceptive Drug Study reported that in a cohort of approximately 16,700 women, the risk of subarachnoid hemorrhage for smokers was 5.7 times that of nonsmokers; the risk for oral contraceptive users was 6.5 times that of nonusers; and the relative risk for women who used both cigarettes and oral contraceptives was 22 times as great. Past users of oral contraceptives also had an increase in relative risk, but an analysis of risk was not possible because of the small number of cases (31).

The risk of myocardial infarction in women is increased by cigarette smoking and by the use of oral contraceptives; it is compounded when both are used together. For example, Mann and associates reported a retrospective study of 63 women below the age of 45 with acute myocardial infarction. The proportion of heart attack patients who had used oral contraceptives in the previous months was significantly higher than expected. The relative risk for myocardial infarction among women smoking 25 or more cigarettes per day was 11.3 times greater than that among nonsmokers. Moreover, there was evidence for synergism of the two risks (23).

Jick, et al. reported a case control study of 107 women under age 46 who were discharged from the hospital after suffering nonfatal, acute myocardial infarctions (15,16,17). The annual risk of nonfatal myocardial infarction (MI) among healthy women aged 39 to 45 who both smoked and used estrogens for noncontraceptive purposes was approximately 1 in 750. They noted that although an acute myocardial infarction is uncommon in healthy young women, the risk appears to be substantial in women over the age of 38 who both use estrogens and smoke cigarettes (17).

In this same study, a relative risk of 14 was reported for oral contraceptive users compared with nonusers (90 percent confidence limits of relative risk from 5.5 to 37) (16). In women smoking more than 25 cigarettes per day the relative risk rose to 34 times that of women who were both nonusers and nonsmokers. While the number of subjects was small, the authors calculated that for women exposed to either oral contraceptives or smoking, but not both, the annual age-specific risks for nonfatal MI were roughly 1 per 190,000 at ages 27 to 37; 1 per 47,000 at ages 38 to 40; 1 per 23,000 at ages 40 to 43; and 1 per 16,000 at ages 44 and 45. If, however, both cigarettes and oral contraceptives are used, the annual age-specific risk is estimated to be much higher and the respective risks become 1 in 8,400; 1 in 920, 1 in 540, and 1 in 250. The authors report that a dose-response relationship exists between smoking and risk among their population of female myocardial infarction patients, such that smoking 1 to 14 cigarettes per day carried a relative risk of nonfatal myocardial infarction of 9.2; 15 to 25 cigarettes of 7.9; and 26 or more cigarettes of 21, relative to those who never smoked (15).

In another recent study of 234 pre-menopausal women who had suffered a first myocardial infarction and 1,742 control patients drawn from the hospital population, Shapiro and his coworkers found an association between recent oral contraceptive use and smoking (35). They found no evidence that past use of oral contraceptives was related to heart attack or that heightened risk was associated with increased duration of use of the oral contraceptives. For nonsmokers who used oral contraceptives, the rate of myocardial infarction increased fourfold compared to nonusers and nonsmokers; in those women who smoked 25 or more cigarettes a day but did not use oral contraceptives, the rate increased more than sevenfold; and in those women who both smoked heavily and used oral contraceptives the rate increased at least twentyfold.

Carbon Monoxide

A study of male and female office workers found no sex difference in the relationship between carboxyhemoglobin (COHb) levels and daily consumption of cigarettes. However, women smoked fewer cigarettes on the average than men. The study found that the COHb levels in smokers were higher among the sedentary office workers than among physically active meat porters and that both had higher levels of COHb than pregnant women who smoked (12). The latter had COHb levels approximately three times higher than that of nonsmokers. Wald reported from a cross-sectional study that carboxyhemoglobin levels of smokers are a better indicator of the risk of atherosclerotic cardiovascular disease than a reported smoking history (48). The proportion of both men and women with atherosclerotic disease increased with increasing levels of COHb.

Comment

Women are less likely to experience a myocardial infarction than men. Nevertheless, coronary heart disease is still a leading cause of death and disability in women. The lower mortality rates from acute myocardial infarction and chronic ischemic heart disease of women as compared to men are paralleled by less extensive and severe atherosclerosis in the coronary artieries of adult women. The severity of aortic atherosclerosis, however, is about the same in both sexes.

The relationship of cigarette smoking to atherosclerosis, heart attack,and other ischemic diseases secondary to atherosclerosis has not been studied among women as extensively as among men; moreover, most studies have been limited to white women. It is not known whether atherosclerotic plaques observed at autopsy are more extensive and severe in women smokers than in nonsmokers. No data are available concerning the incidence of death from atherosclerotic aneurysms of the aorta among women who smoke relative to those who do not, and inadequate data exist to indicate whether cessation of smoking by women is associated with a beneficial reduction in the risk of heart attack, as has been demonstrated in men. The effect of smoking on the threshold for the onset of angina pectoris and on cardiac function in women with coronary heart disease has not been studied.

Nevertheless, compelling data from prospective cohort studies and from case control investigations indicate that cigarette smoking is a major risk factor for fatal and nonfatal heart attacks in women. In general, cigarette smoking increases the risk by a factor of about two, and in younger women cigarette smoking may increase the risk several fold. Women who smoke low-"tar" and low-nicotine cigarettes have a greater risk of suffering heart attacks than nonsmokers but appear to have a smaller risk than women smoking moderate-to-high "tar" and nicotine products.

Smoking is a major risk factor for arteriosclerotic peripheral vascular disease in women, as it is in men. For both men and women the successful outcome of surgical repair of this disorder is enhanced by cessation of smoking. Smoking is a major risk factor for subarachnoid hemorrhage and for the development of malignant hypertension. Smoking is reported to depress the natural relative elevation of high-density lipoprotein cholesterol enjoyed by women. In women who use oral contraceptives, smoking is a powerful synergistic risk factor for subarachnoid hemorrhage and for myocardial infarction.

While data implicating smoking as a risk factor for various cardiovascular diseases in women are neither as extensive nor as complete as for men, the evidence nonetheless clearly establishes cigarette smoking as a major correlate for myocardial infarction, arteriosclerotic peripheral vascular disease and subarachnoid hemorrhage in women (45).

Summary

Coronary heart disease is the major cause of death among both males and females in the U.S. population. The 1979 Surgeon General's Report clearly demonstrated the close association of cigarette smoking and increased coronary heart disease among males. This report reviews the evidence associating cigarette smoking and cardiovascular disease in women:

1. Coronary heart disease, including acute myocardial infarction and chronic ischemic heart disease, occurs more frequently in women who smoke. In general, cigarette smoking increases the risk by a factor of about two, and in younger women cigarette smoking may increase the risk several fold.
2. Cigarette smoking is a major independent risk factor for coronary heart disease in women; it also acts synergistically with other coronary heart disease risk factors producing a risk greater than the sum of the individual risks.
3. The use of oral contraceptives by women cigarette smokers

increases the risk of a myocardial infarction by a factor of approximately ten.

4. Women who smoke low "tar" and nicotine cigarettes experience less risk for coronary heart disease than women who smoke high "tar" and nicotine cigarettes, but their risk is still considerably greater than that of nonsmokers.

5. Increased levels of high-density lipoprotein (HDL) are correlated with a reduced risk for an acute myocardial infarction; women cigarette smokers have decreased levels of HDL.

6. Cigarette smoking is a major, independent risk factor for the development of arteriosclerotic peripheral vascular disease in women. Smoking cessation improves the prognosis of the disorder and has a favorable impact on vascular patency following reconstructive surgery.

7. Women cigarette smokers experience an increased risk for subarachnoid hemorrhage; the use of both cigarettes and oral contraceptives appears to increase synergistically the risk for subarachnoid hemorrhage.

8. Women who smoke cigarettes may be more likely to develop severe or malignant hypertension than nonsmoking women.

References

(1) ARNTZENIUS, A.C., VAN GENT, C.M., VAN DER VOORT, H., STEGERHOEK, C.I., STYBLO, K. Reduced high-density lipoprotein in women aged 40–41 using contraceptives. Consultation Bureau Heart Project. Lancet: 1221–1223, June 10, 1978.

(2) BELL, B.A., SYMON, L. Smoking and subarachnoid haemorrhage. British Medical Journal 1: 577–578, March 3, 1979.

(3) BLOXHAN, C.A., BEEVERS, D.G., WALKER, J.M. Malignant hypertension and cigarette smoking. British Medical Journal 1:581–583, March 3, 1979.

(4) BRADLEY, D.D., WINGERD, J., PETITTI, D.B., KRAUSS, R.M., RAMCHARAN, S. Serum high-density lipoprotein cholesterol in women using oral contraceptives, estrogens, and progestins. New England Journal of Medicine 299(1): 17–20, July 6, 1978.

(5) COLLABORATIVE GROUP FOR THE STUDY OF STROKE IN YOUNG WOMEN. Oral contraceptive and increased risk of cerebral ischemia or thrombosis. New England Journal of Medicine 288(17): 871–878, April 26, 1973.

(6) DOLL, R., et al. Mortality in relation to smoking: 20 years' observations on male British doctors. British Medical Journal 2: 1525–1536, December 25, 1976.

(7) DOLL, R., GRAY, R., PETO, R. Mortality in Relation to Smoking: Observations on Female Doctors. (Unpublished manuscript)

(8) EPSTEIN, F.H. Some uses of prospective observations in the Tecumseh Community Health Study. Proceedings of the Royal Society of Medicine 60(1): 56–60, January 1967.

(9) GARRISON, R.J., KANNEL, W.B., FEINLEIB, M., CASTELLI, W.P., MCNAMARA, P.M., PADGETT, S.J. Cigarette smoking and

HDL cholesterol: the Framingham Offspring Study. Atherosclerosis 30: 17-25, 1978.

(10) HAMMOND, E.C., GARFINKEL, L. Coronary heart disease, stroke, and aortic aneurysm: factors in the etiology. Archives of Environmental Health 19(2): 167-182, August 1969.

(11) HAMMOND, E.C., GARFINKEL, L., SEIDMAN, H., LEW, E.A. "Tar" and nicotine content of cigarette smoke in relation to death rates. Environmental Research 12(3): 263-274, December 1976.

(12) HAWKINS, L.H. Blood carbon monoxide levels as a function of daily cigarette consumption and physical activity. British Journal of Industrial Medicine 33(2): 123-125, May 1976.

(13) ISLES, C., BROWN, J.J., CUMMING, A.M.M., LEVER, A.F., MCAREAVEY, D., ROBERTSON, J.I.S., HAWTHORNE, V.M., STEWART, G.M., ROBERTSON, J.W.K., WAPSHAW, J. Excess smoking in malignant-phase hypertension. British Medical Journal 1: 579-581, March 3, 1979.

(14) JAIN, A.K. Cigarette smoking, use of oral contraceptives, and myocardial infarction. American Journal of Obstetrics and Gynecology 126(3): 301-307, October 1, 1976.

(15) JICK, H., DINAN, B., HERMAN, R., ROTHMAN, K.J. Myocardial infarction and other vascular diseases in young women. Role of estrogens and other factors. Journal of the American Medical Association 240(23): 2548-2552, December 1, 1978.

(16) JICK, H., DINAN, B., ROTHMAN, K.J. Oral contraceptives and nonfatal myocardial infarction. Journal of the American Medical Association 239(14): 1403-1406, April 3, 1978.

(17) JICK, H., DINAN, B., ROTHMAN, K.J. Noncontraceptive estrogens and non-fatal myocardial infarction. Journal of the American Medical Association 239(14): 1407-1408, April 3, 1978.

(18) KAGAN, A.R., STERNBY, N.H., UEMURA, K., VANECEK, R., VIHERT, A.M., LIFSIC, A.M., MATOVA, E.E., ZAHOR, Z., ZDANOV, V.S. Atherosclerosis of the aorta and coronary arteries in five towns. Bulletin of the World Health Organization 53(5-6): 501-530, 1976.

(19) KANNEL, W.B. Epidemiologic studies on smoking in cerebral and peripheral vascular disease. In: Wynder, E.L., Hoffmann, D., Gori, G.B. (Editors). Proceedings of the Third World Conference on Smoking and Health, New York, June 2-5, 1975. Volume I. Modifying the Risk for the Smoker. U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute, DHEW Publication No. (NIH) 76-1221, 1976, pp. 257-274.

(20) KANNEL, W.B., CASTELLI, W.P., MCNAMARA, P.M. Cigarette smoking and risk of coronary heart disease. Epidemiologic clues to pathogenesis. The Framingham Study. In: Wynder, E.L., Hoffmann, D. (Editors). Toward a Less Harmful Cigarette. National Cancer Institute Monograph No. 28. U.S. Department of Health, Education, and Welfare, Public Health Service, National Cancer Institute, June 1968, pp. 9-20.

(21) KURTZKE, J.F. Epidemiology of cerebrovascular disease. In: Cerebrovascular survey report for the Joint Council Subcommittee on Cerebrovascular Disease. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, January 1976, pp. 213-242.

(22) LAWSON, D.H., DAVIDSON, J.F., JICK, H. Oral contraceptive use and venous thromboembolism: abscence of an effect of smoking. British Medical Journal 2: 729-730, September 17, 1977.

(23) MANN, J.I., VESSEY, M.P., THOROGOOD, M., DOLL, R. Myocardial infarction in young women with special reference to oral contraceptive practice. British Medical Journal 2: 241–245, May 3, 1975.

(24) MCGILL, H.C., JR. (Editor). The geographic pathology of atherosclerosis. General Findings of the International Atherosclerosis Project. Laboratory Investigation 18(5): 498–502, 1968.

(25) MCGILL, H.C., JR., STERN, M.P. Sex and Atherosclerosis. In: Paoletti, R., Gotto, A.M., Jr. (Editors). Atherosclerosis Reviews. Volume 4. New York, Raven Press, 1979, pp. 157–242.

(26) MEADE, T.W., NORTH, W.R.S., CHAKRABARTI, R., HAINES, A.P., STIRLING, Y. Population-based distributions of haemostatic variables. British Medical Bulletin 33(3): 283–288, 1977.

(27) MYERS, K.A., KING, R.B., SCOTT, D.F., JOHNSON, N., MORRIS, P.J. The effect of smoking on the late patency of arterial reconstructions in the legs. British Journal of Surgery 65(4): 267–271, April 1978.

(28) NAEYE, R.L., TRUONG, L.D. Effects of cigarette smoking on intramyocardial arteries and arterioles in man. American Journal of Clinical Pathology 68(4): 493–498, October 1977.

(29) NAKAYAMA, Y. Epidemiological research in Japan on smoking and cardiovascular diseases. In: Schettler, G., Goto, Y., Hata, Y., Klose, G. (Editors). Atherosclerosis IV. Proceedings of the Fourth International Symposium, Tokyo, 1976. Berlin, Springer-Verlag, 1977, pp. 149–153.

(30) OMAE, T., TAKESHITA, M., HIROTA, Y. The Hisayama study and joint study on cerebrovascular diseases in Japan. In: Scheinberg, P. (Editor). Cerebrovascular Diseases. Proceedings of the Tenth Princeton Conference, New Jersey, 1976. New York, Raven Press, 1976, pp. 255–265.

(31) PETITTI, D.B., WINGERD, J. Use of oral contraceptives, cigarette smoking, and risk of subarachnoid haemorrhage. Lancet 2: 234–236, July 29, 1978.

(32) PETITTI, D.B., WINGERD, J., PELLEGRIN, F., RAMCHARAN, S. Oral contraceptives, smoking, and other factors in relation to risk of venous thromboembolic disease. American Journal of Epidemiology 108(6): 480–485, December 1978.

(33) ROSENBERG, H.M., KLEBBA, A.J. In: Havlick, R.J., Feinleib, M. (Editors). Proceedings of the Conference on the Decline in Coronary Heart Disease Mortality. Department of Health, Education, and Welfare, Public Health Service, NIH Publication No. 79-1610, 1979, pp. 11–39.

(34) SACKETT, D.L., GIBSON, R.W., BROSS, I.D.J., PICKREN, J.W. Relation between aortic atherosclerosis and the use of cigarettes and alcohol. An autopsy study. New England Journal of Medicine 279(26): 1413–1420, December 26, 1968.

(35) SHAPIRO, S., SLONE, D., ROSENBERG, L., KAUFMAN, D.W., STOLLEY, P.D., MIETTINEN, O.S. Oral-contraceptive use in relation to myocardial infarction. Lancet 1: 743–747, April 7, 1979.

(36) SHAPIRO, S., WEINBLATT, E., FRANK, C.W., SAGER, R.V. Incidence of coronary heart disease in a population insured for medical care (HIP). Myocardial infarction, angina pectoris, and possible myocardial infarction. American Journal of Public Health 59 (Supplement 6): 1–101, June 1969.

(37) SLONE, D., SHAPIRO, S., ROSENBERG, L., KAUFMAN, D.W., HARTZ, S.C., ROSSI, A.C., STOLLEY, P.D., MIETTINEN, O.S. Relation of cigarette smoking to myocardial infarction in young women. New England Journal of Medicine 298(23): 1273–1276, June 8, 1978.

(38) SPAIN, D.M., SIEGEL, H., BRADESS, V.A. Women smokers and sudden

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death. The relationship of cigarette smoking to coronary disease. Journal of the American Medical Association 224(7): 1005–1007, May 14, 1973.

(39) STAMLER, J., RHOMBERG, P., SCHOENBERGER, J.A., SHEKELLE, R.B., DYER, A., SHEKELLE, S., STAMLER, R., WANNAMAKER, J. Multivariate analysis of the relationship of seven variables to blood pressure. Findings of the Chicago Heart Association Detection Project in Industry, 1967–1972. Journal of Chronic Diseases 28(10): 527–548, November 1975.

(40) STERNBY, N.H. Atherosclerosis in a defined population. An autopsy survey in Malmo, Sweden. Acta Pathologica et Microbiologica Scandinavica. Supplement 194: 16–194, 1968.

(41) STRONG, J.P., SOLBERG, L.A., RESTREPO, C. Atherosclerosis in persons with coronary heart disease. Laboratory Investigation 18(5): 527–537, May 1968.

(42) TALBOTT, E., KULLER, L.H., DETRE, K., PERPER, J. Biologic and psychosocial risk factors of sudden death from coronary disease in white women. American Journal of Cardiology 39(6): 858–864, May 26, 1977.

(43) THE EPIDEMIOLOGY STUDY GROUP. Epidemiology for stroke facilities planning. Report of the Joint Committee for Stroke Facilities. Stroke 3: 360–371, May–June 1972.

(44) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking. A Reference Edition. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, DHEW Publication No. (CDC) 78-8357, 1976, 657 pp.

(45) U.S. PUBLIC HEALTH SERVICE. Smoking and Health. A Report of the Surgeon General. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, DHEW Publication No. (PHS) 79-50066, 1979, 1241 pp.

(46) VESSEY, M.P., DOLL, R. Investigation of relation between use of oral contraceptives and thromboembolic disease. A further report. British Medical Journal 2(5658): 651–657, June 14, 1969.

(47) VESSEY, M., DOLL, R., PETO, R., JOHNSON, B., WIGGINS, P. A long-term follow-up study of women using different methods of contraception. An interim report. Journal of Biosocial Sciences 8: 373–427, 1976.

(48) WALD, N., HOWARD, S., SMITH, P.G., KJELDSEN, K. Association between atherosclerotic diseases and carboxyhaemoglobin levels in tobacco smokers. British Medical Journal 1: 761–765, March 31, 1973.

(49) WEISS, N.S. Cigarette smoking and arteriosclerosis obliterans: an epidemiologic approach. American Journal of Epidemiology 95(1): 17–25, 1972.

(50) WILHELMSEN, L. Recent studies on smoking and CVD epidemiology: Scandinavia and some other Western European countries. In: Steinfeld, J., Griffiths, W., Ball, K., Taylor, R.M. (Editors). Proceedings of the Third World Conference on Smoking and Health, New York, June 2–5, 1975. Volume II. Health Consequences, Education, Cessation Activities and Social Action. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute, DHEW Publication No. (NIH) 77-1413, 1977, pp. 171–177.

CANCER

Introduction

For more than 40 years cancer has been second only to cardiovascular disease as a cause of death in the United States. With the exception of the very elderly, the death rate for adult men exceeds that for adult women for both groups of diseases, implying a difference in genetic susceptibility, environmental exposures or lifestyles between the sexes, or a combination of genetic and environmental factors.

Placing these generalizations about cause of death in perspective, current data from the National Center for Health Statistics (28) reveal the following statistics:

There are 105 male births each year in the United States for every 100 female births, but the higher death rate for males results in a ratio of 100 men to 100 women at ages 20 to 24 and of 79:100 at ages 65 to 69, and of 47:100 at age 85. Life expectancy in the United States in 1976 was 68.7 years for males compared to 76.1 years for females.

Heart disease and cancer currently account for 60 percent of deaths in the United States. In contrast to the decline in the age-adjusted death rates for ischemic heart disease, the age-adjusted death rate for cancer has increased. Hidden in this small rise in the overall cancer statistics is a remarkable increase—a veritable epidemic—of cancer of the lung in both men and women. In the past quarter century, deaths from cancer of the respiratory tract tripled in the white population and quadrupled in the black population. The remarkable male-to-female preponderence of lung cancer in the 1940s and 1950s has been decreasing in the 1960s and 1970s; the rate of increase in lung cancer in males is slowing while the rate of increase of lung cancer in females is accelerating. As a cause of death, lung cancer in women is now second only to mammary carcinoma and will likely displace breast cancer as the leading cause of cancer mortality in women in the 1980s (1) (see Figure 1).

The 1964 Surgeon General's Report reached the following conclusion: "Cigarette smoking is causally related to lung cancer in men; the magnitude of the effects of cigarette smoking far outweighs all other factors. The data for women, though less extensive, point in the same direction" (33). Since then, a number of retrospective and prospective epidemiologic studies, experimental animal carcinogenesis studies, and studies of human tissues at surgery and autopsy have confirmed and extended those conclusions. Cigarette smoking is the major cause of cancer of the lung in women. The risk increases with the number of years the individual smoked, the number of ciga-

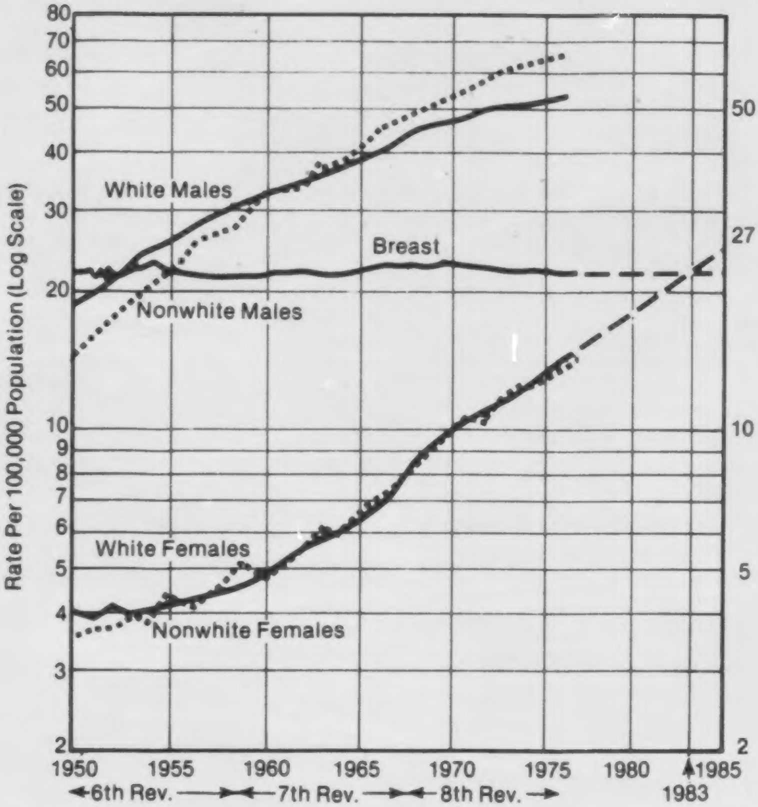

FIGURE 1.—Age-adjusted death rates* for malignant neoplasm of trachea, bronchus and lung,** by color and sex compared to rates for malignant breast neoplasm, United States, 1950–1977; projection for white females to 1985.***

*Adjusted by the direct method to the U.S. population, 1940.
**ICD 6th and 7th Rev. Nos. 162, 163 and 8th Rev. No. 162.
***Projection based on average annual rate of increase over last 10 years.
SOURCE: National Cancer Institute (25), National Center for Health Statistics (27).

rettes smoked, the "tar" and nicotine level of the cigarette smoked and the degree of inhalation, and is inversely related to the age at which the individual began smoking, being higher for those who begin smoking at younger ages. The risk of developing cancer is diminished significantly by quitting smoking and is lessened somewhat by switching to low-tar, low-nicotine filter-tip cigarettes (43,45). Considerable evidence has also shown that cigarette smoking is a significant cause—for women and men—of cancer of the larynx, oral cavity, esophagus, urinary bladder, kidney, and pancreas. Much of this information has been summarized in previous issues of "The Health Consequences of Smoking" or the Surgeon General's Reports (33–43).

Table 1 lists the new cases and deaths estimated to occur in 1980 for those cancers which are causally associated with cigarette smoking (1). Smoking will contribute to 43 percent of the male and 18 percent of the female newly diagnosed cancer cases in the United States in 1980 and to 51 percent of the male and 26 percent of the female cancer deaths. This table does not imply that cigarette smoking causes each of these individual cancers. It does, however, identify the impact of cigarette smoking on the major cancers now known to be associated with cigarette smoking. Most of the cases of cancer of the lung and larynx could have been prevented, as could a substantial proportion of the cancer deaths at the other sites listed.

In this chapter, selected data on cancer and smoking among women will be reviewed and summarized. Where necessary for clarity, data previously reported will be summarized briefly.

Lung

The lung is a complex organ lined by at least five types of epithelial cells, each of which theoretically might give rise to one or more types of neoplasm. In addition to the epithelial cells, blood vessels and connective tissue are prominent in the lungs. Both visceral and parietal portions of the lung are covered by synovial membranes, which also are subject to neoplastic transformation. The World Health Organization's classification of malignant tumors (Table 2) includes multiple histologic types, of which epidermoid, small cell, adenocarcinoma, and large cell carcinoma are causally related to cigarette smoking and display significant dose-response relationships in epidemiologic studies (7,43). These four tumors are the most common histologic types of lung cancer in both men and women. However, there are differences in the distribution of the different types of lung cancer in men and women and in smokers and nonsmokers. Epidermoid carcinoma was the most common histologic type of lung cancer in the male smoker, while adenocarcinoma was most common in the female smoker and in nonsmokers of both sexes in a series recently published from the Mayo Clinic (Table 3) (31).

TABLE 1.—Estimated new cancer cases and deaths for sites associated with cigarette smoking, 1980

Site	Estimated New Cases			Estimated Deaths		
	Total	Male	Female	Total	Male	Female
All Sites	785,000*	387,000*	398,000*	405,000	219,500	185,500
Lung	117,000	85,000	32,000	101,300	74,800	26,500
Pancreas	24,000	12,500	11,500	20,900	11,100	9,800
Urinary Bladder	35,500	26,000	9,500	10,300	7,000	3,300
Oral	25,500	17,900	7,600	8,800	6,100	2,700
Kidney & Other Urinary	16,900	10,500	6,400	7,900	4,800	3,100
Esophagus	8,800	6,200	2,600	7,600	5,500	2,100
Larynx	10,700	9,000	1,700	3,500	2,900	600
All Tobacco Related	238,400	167,100	71,300	160,300	112,200	48,100

*Carcinoma *in situ* is not included. There are 45,000 new cases of uterine cervical carcinoma *in situ* each year. Non-melanoma skin cancer is not included. Approximately 400,000 new cases of non-melanoma skin cancer occur annually.
SOURCE: American Cancer Society (1).

TABLE 2.—World Health Organization classification of malignant pleuro-pulmonary neoplasms

I. Epidermoid Carcinomas
II. Small Cell Anaplastic Carcinomas
III. Adenocarcinomas
  1. Bronchogenic
    a. acinar
    b. papillary with or without mucin formation
IV. Large Cell Carcinomas
V. Combined Epidermoid and Adenocarcinomas
VI. Carcinoid Tumors
VII. Bronchial Gland Tumors
  1. Cylindromas
  2. Mucoepidermoid tumors
VIII. Papillary Tumors of the Surface Epithelium
IX. Mixed Tumors and Carinosarcomas
X. Sarcomas
XI. Unclassified
XII. Melanoma
XIII. Mesotheliomas

SOURCE: Kreyberg, L. (22).

TABLE 3.—Histologic types of pulmonary cancers in smokers and nonsmokers

		Male		Female	
Type	Total	Smokers	Non-Smokers	Smokers	Non-Smokers
Epidermoid	992	892	7	80	13
Small Cell	640	533	4	100	3
Adenocarcinoma	760	492	39	128	101
Large Cell	466	389	16	46	15
Bronchioloalveolar	68	35	4	13	16
TOTAL	2,926	2,341	70	367	148

SOURCE: Resenow, E.C. (31).

Other centers have similar data, although the proportions by histologic type may vary with the pathologic criteria used, patient population, geographic location, and other factors.

Earlier epidemiologic studies suggested that cigarette smokers were more likely to develop squamous-cell and small-cell lung carcinoma than other types. However, more recent investigations indicate that all four major histologic types of lung cancer—including adenocarcinoma, which appears to be increasing rapidly in recent years—are related to cigarette smoking in both men and women (43).

In 1980, of the estimated 117,000 newly diagnosed cancers of the lung in the United States, 32,000 will be among women. There will be an estimated 25,500 deaths from lung cancer in women (1).

In 1950, women accounted for approximately 1 in 12 of all lung cancer deaths. By 1968 the proportion was 1 in 6; in 1979 women dying of lung cancer will represent over one-quarter of all lung cancer victims. White women have death rates from lung cancer which are similar to those of nonwhite women, while the rates of white males remain below those of nonwhite males. These differences may be due to differences in the smoking habits of blacks and whites described elsewhere in this report.

Many prospective studies have found that the lung cancer death rate for smokers was far in excess of the rates for nonsmokers in both sexes; as previously mentioned, the rates for male smokers dramatically exceeded the rates for female smokers. However, even the nonsmoking male had a higher incidence of, and death rate from, lung cancer than the nonsmoking female (9). This evidence suggested that women might have a decreased susceptibility to lung cancer. A more careful examination of the data indicates that most of the differences between male and female lung cancer rates can be explained by differences in smoking habits and occupational exposures.

As discussed in other sections of this report, a smaller percentage of women than men smoke and, when they do smoke, they are more likely to adopt smoking behaviors that have been shown to have a lower risk of developing lung cancer. That is, they smoke fewer cigarettes per day, inhale less, start smoking later in life, and are more likely to smoke low-tar and low-nicotine and filter cigarettes. In addition, it is important to consider the cohort effects on the differences in rates between males and females. Over 85 percent of those who smoke regularly began between the ages of 12 and 25 (29). Men first began to smoke in large numbers just before and during the First World War. As each succeeding birth cohort passed through the age of initiation (12 to 25), a larger percentage began smoking until the groups born between 1915 and 1930 were reached (17). In the birth cohorts born after 1930, fewer began to smoke regularly. The risk of developing lung cancer increases exponentially with age and duration of smoking, with the increase starting 15 to 20 years after the beginning of regular smoking. This accounts for the dramatic rise in the male lung cancer death rates noted in the 1930s. As those birth cohorts with higher smoking rates replaced those with lower smoking rates, the age-specific lung cancer rates rose steadily; and as each of the heavy-smoking birth cohorts grew older, their lung cancer risk continued to accelerate, resulting in a very steep rise in the overall male lung cancer death rate. The overall cancer rates among men will continue to rise (albeit more slowly) as those birth cohorts with the heaviest smoking prevalence replace those with lower prevalence in the older age groups where the lung cancer death rates are the highest. As these birth cohorts with high smoking prevalence pass through the age groups and are replaced by birth cohorts with lower smoking prevalence, declines in lung cancer rates should be noted.

They should be noted first in the age-specific death rates for the younger age groups and later in the overall lung cancer death rates. The first indications of this change have been noted with a decline in the age-specific death rates in males born after 1930. It is therefore important to consider this cohort effect when examining the differences between lung cancer rates of men and women.

Women began to take up smoking in large numbers 20 to 30 years later than men (in the early 1940s). This rise in smoking prevalence was produced by predominantly young women first using tobacco as cigarettes. This is in contrast to the rise in men which included a substantial percentage of men of all ages who switched from other forms of tobacco use to cigarettes. The rise in lung cancer rates in women occurred as those cohorts with high smoking prevalence reached the ages where lung cancer occurs with significant frequency (age 45 and over). Since most of these women began smoking cigarettes prior to age 25 they would have at least 20 years of exposure by age 45 in contrast to the shorter durations of exposure at age 45 for those men who switched to cigarettes from other forms of tobacco around the time cigarettes first came into widespread use. This greater duration of exposure at any given age for women in these first heavy smoking birth cohorts compared to the first cohorts in men, should result in a more abrupt rise in lung cancer rates in women. This rapid rise in female lung cancer death rates began to be observed in the late 1950s. As birth cohorts with higher smoking prevalence continued to replace those with lower smoking prevalence, the rates rose steeply, reproducing the phenomenon noted in males 20 to 30 years earlier with some indication that the rise is even steeper for women. If one subtracts 25 years from the female cancer death rates in Figure 1, the rates for women are only slightly below the rates for men. This small difference is explained by lower prevalence of smoking and less hazardous smoking patterns of women and their less frequent exposure to occupational carcinogens. Thus, close scrutiny of the trends reveals no substantial protective effect for women on the risk of developing lung cancer but rather leads to a

TABLE 4.—Age-adjusted lung cancer mortality ratios—age began smoking and degree of inhalation

Age Began Smoking	Male	Female
15	16.8	2.5
15-19	14.7	5.0
20-24	10.1	3.4
25+	4.1	2.3
Depth of Inhalation	**Male**	**Female**
None	8.0	2.0
Slight	8.9	2.3
Moderate	13.1	3.5
Heavy	17.0	7.1

SOURCE: Hammond, E.C. (11).

TABLE 5.—Age-adjusted relative risks of lung cancer by number of cigarettes smoked

		Number of Cigarettes Smoked Daily			
		1-9	10-19	20-39	40+
ACS Study	Male	4.6	8.6	14.7	18.8
	Female	1.3	2.4	4.9	7.5
		1-14		15-24	25+
British Physicians	Male	7.8		12.7	25.1
	Female	1.3		6.4	29.7

SOURCE: Doll, R. (6,8), Hammond, E.C. (11).

sobering projection of a reproduction of the male lung cancer epidemic in women (Figure 1).

GEOGRAPHIC DIFFERENCES

Lung cancer death rates, including all histologic types, are highest in industrialized countries where there has been a higher smoking prevalence for a longer time. Women in Scotland have one of the highest death rates from lung cancer of women of any country. Their tobacco consumption per smoker approaches that of English and Welsh men (19). Current tobacco consumption by Scottish women is only a little lower than the consumption of Scottish men 20 years ago. In England and Scotland, where the upper socioeconomic classes have reduced their

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TABLE 6.—Lung cancer mortality ratios for females by duration of smoking: Swedish study

Duration of Smoking in Years	Mortality Ratios
Nonsmokers	1.0
1–29 years	1.6
30+ years	9.6

SOURCE: Cederlof, R. (4).

cigarette consumption in recent decades, there is a significantly greater lung cancer mortality rate in the lower socioeconomic classes among women (19).

Age-adjusted death rates for lung cancer in women in select countries indicate that women in Hong Kong have the highest rates, while those in Scotland are second and those in England and Wales are third. The United States ranked sixth world wide (1).

Among nonsmokers, lung cancer is found slightly more often in urban than in rural areas; however, the marked increase in lung cancer among smokers in urban areas suggests that urban living exerts a potentiating rather than an additive effect on the incidence of lung cancer. Urban living has little independent effect on lung cancer induction in comparison with even modest smoking of filtered low-tar and low-nicotine cigarettes (5,10).

SMOKING PATTERNS AMONG WOMEN

Although women tend to have different patterns of smoking than men, the relative relationships between smoking and lung cancer are the same. Lung cancer rates for women who smoke increase with increased dosage as measured by several dosage measures, including number of cigarettes smoked per day, duration of smoking habit, degree of inhalation, age of initiation of smoking, and the "tar" and nicotine level of the cigarettes smoked. These data, obtained from several prospective investigations, are examined in Tables 4, 5, 6, 7, 9, and 10. The more cigarettes an individual smokes, the more likely that individual will die of lung cancer (Table 5). Overall, female cigarette smokers have 2.5 to 5.0 times greater likelihood of dying from lung cancer than nonsmokers (Table 7). As discussed earlier, when the full impact of the cohort effect is felt, this ratio will probably approach that for men (8 to 12).

Doll, et al. studied the cause-specific mortality experience among approximately 6,200 female physicians in England during

TABLE 7.—Lung cancer mortality prospective studies

Age Adjusted Lung Cancer Death—Relative Risks			
		Nonsmokers	Cigarette Smokers
ACS	Male	1.0	10.1
	Female	1.0	2.6
British Physicians	Male	1.0	14.0
	Female	1.0	5.0
Swedish Study	Male	1.0	8.2
	Female	1.0	4.5

SOURCE: Cederlof, R. (4), Doll, R. (6,8), Hammond, E.C. (11).

the period 1951 to 1973 (6). The results of this study are presented in detail in Table 8, which also includes data from a previous report on male physicians (8).

It is apparent that smoking and lung cancer are similarly related in men and women. In both sexes, lung cancer mortality was at least three times as high in ever-smokers as in never-smokers, at least twice as high in current heavy smokers (more than 25 cigarettes) as in light smokers (less than 15 cigarettes), and exhibited a significant dose-response relationship. The magnitude of the smoking effect on lung cancer for females and males was approximately the same. The relative risks for mortality from lung cancer for moderate (15 to 24 cigarettes per day) and heavy (more than 25 cigarettes) smokers were 6.3 and 29.7 among females, and 10.6 and 22.4 for males.

The authors emphasize, however, that no conclusions can be drawn from this data about the magnitude of the biologic effects of smoking in men compared to women. Since the authors documented differences in lifetime smoke exposure (later age at initiation and lower prevalence of inhalation among females), lifetime smoking exposures between the sexes were not directly comparable. This issue will be resolved only when studies examine the effect of smoking in cohorts of women whose lifetime smoking behavior more closely matches that of the men to whom they are compared.

A number of retrospective studies have examined the relationship of smoking and lung cancer in women. The 1971 Health Consequences of Smoking reviewed many of these investigations and showed a smoker-to-nonsmoker risk ratio ranging from 0.2 to 6.8 for females. The reader is referred to this volume for a more detailed discussion of these studies. Results of these investigations reveal sex differentials similar to those found in

TABLE 8.—Death rates from lung cancer and smoking habit when last asked, British physicians 1951–1973

			Annual Death Rate per 100,000 Persons Standardized for Age					X^2	
					Current Smokers—Dose Per Day				
	Total Popul.	# Deaths	Nonsmokers	Ex-Smokers	1–14	15–25	25+	Nonsmokers vs. Others	Trend (Dose/ Response)
Women	6,194	27	7	23	9	45 (cigarettes only)	208	13.47*	61.59*
Men	34,440	441	10	43	52	106 (any tobacco/grams) (1 gram = 1 cigarette)	224	41.9*	197.04*

*(P<.001)
SOURCE: Doll, R. (6,8).

TABLE 9.—Age-adjusted lung cancer mortality ratios* for males and females, by tar and nicotine (T/N) in cigarettes smoked

	Males	Females
High T/N	1.00	1.00
Medium T/N	0.95	0.79
Low T/N	0.81	0.60

*The mortality ratio for the category with highest risk was made 1.00 so that the relative reductions in risk with the use of lower T/N cigarettes could be visualized.

SOURCE: Hammond, E.C. (11).

the larger prospective studies, with males having higher overall lung cancer rates compared to females. However, the lung cancer rates of smokers are significantly higher than those of nonsmokers for both sexes.

The women who smoke low-"tar", low-nicotine cigarettes have a lower age-adjusted lung cancer mortality rate than women who smoke high-"tar", high-nicotine cigarettes. Women who smoke medium-"tar", medium-nicotine cigarettes have mortality rates in between (12) (Table 9). However, even the low-"tar" and low-nicotine cigarette smoker has a rate substantially higher than the nonsmoker.

These data suggest some benefit from smoking low-"tar", low-nicotine cigarettes. However, a further comparison of women who smoked less than one pack of high-"tar", high-nicotine cigarettes daily with women who smoked more than one pack of low-"tar", low-nicotine cigarettes daily revealed that the smoker of more than a pack a day of low-"tar", low-nicotine cigarettes had over twice the age-adjusted lung cancer mortality rate of the woman who smoked fewer cigarettes, but with high "tar" and nicotine (Table 10).

In a retrospective study standardized for duration of smoking, number of cigarettes smoked, inhalation and butt length, long-term female smokers of filter cigarettes had a lower relative risk of developing cancer than smokers of non-filter cigarettes (46).

CESSATION OF SMOKING

Although the risk of developing lung cancer increases with age, both for smokers and nonsmokers alike, women in good health who quit smoking will, over a period of years, experience a reduction in their relative risk of developing lung cancer. About 15 years after they have quit smoking, the risk of developing lung cancer approximates that of the nonsmoker.

TABLE 10.—Age-adjusted lung cancer mortality ratios* for males and females, comparing those who smoked a few high tar and nicotine (T/N) cigarettes with those who smoked many low T/N cigarettes

	1–19 high T/N cigarettes/day	20–39 low T/N cigarettes/day
Males	1.00	1.6
Females	1.00	2.1

*The mortality ratio for the category with lowest risk was made 1.00 so the increase in risk with smoking more cigarettes/day could be illustrated.
SOURCE: Hammond, E.C. (11).

EXPERIMENTAL CARCINOGENESIS

Tobacco tars, tobacco smoke, and single or mixtures of chemicals found in tobacco smoke have been used with various species of animals in carcinogenesis experiments involving skin painting, subcutaneous injections, tracheobronchial implantation, and/or instillation and inhalation. Some experiments have reported sex differences in the occurrence of lung tumors following exposure to chromium oxide (26).

However, in a recent monograph on lung cancer, separate reviews on tobacco carcinogenesis, radiation carcinogenesis in the respiratory tract, and experimental models for studies of respiratory tract carcinogenesis did not yield information suggesting that the male lung of any of the species studied was more susceptible than the female lung to carcinogenic action by either tobacco products or radiation (16). The reader is referred to previous Smoking and Health Reports for summaries of experimental tobacco carcinogenesis studies.

Larynx

The larynx is a small, complex structure, which produces speech, controls the flow of air in and out of the lungs, and prevents aspiration during swallowing. In 1980 there will be an estimated 1,700 new cases of laryngeal cancer and 600 deaths from that tumor in U.S. women (Table 1). Laryngeal cancer has occurred predominantly in men, but more and more women are developing laryngeal cancer as their smoking and drinking habits come to approximate those of men. The male-to-female ratio for laryngeal cancer exceeds that of lung cancer. Laryngeal cancer occurs in the fifth, sixth, and seventh decades both in men and women. While the disease is uncommon, its incidence has continued to rise over the past quarter century, especially in women, substantially because of changes in their smoking habits.

Cancer can occur either in the glottis (true cord, 70 percent of cases), or in the subglottic or supraglottic region (false cord, 25 percent of cases). Usually the neoplasm is epidermoid carcinoma when examined histologically. Since a tumor that interferes with speech gives rise to early symptoms, glottic cancers are usually diagnosed at an early stage and are curable in over 60 percent of the cases. When the tumor arises in the subglottic or supraglottic region, interference with phonation or speech may not occur as early as when neoplasm begins on the glottis. The tumor may, therefore, reach a greater size and be accompanied by significant local tissue invasion and destruction as well as metastasis. Patients with tumors discovered when they are still localized in the larynx have approximately an 80 percent cure rate, while advanced lesions have a 33 percent 5-year survival rate.

Laryngeal cancer displays a strong dose-response relationship with smoking, increasing with the number of cigarettes smoked per day, the "tar" and nicotine content of the cigarettes smoked, the depth of inhalation and number of years cigarettes were smoked. The risk of developing laryngeal cancer is inversely related to the age at which smoking began (43). A lower risk for laryngeal cancer has been demonstrated in women who used filtered cigarettes for 10 years or more compared to those who smoked non-filtered cigarettes. Nonetheless, the risk remained well in excess of that experienced by nonsmokers (45).

Excessive use of alcohol by nonsmokers also results in an increased incidence of laryngeal cancer. Heavy drinkers of alcohol—that is, greater than seven ounces of whiskey or its equivalent per day—who also smoke cigarettes have a greater risk of developing laryngeal cancer than if they either smoked or drank to excess alone. There is a synergistic effect of smoking and drinking on laryngeal cancer development (43,44).

When women quit smoking, their relative risk of developing laryngeal cancer decreases until 10 years after cessation when their risk approaches that of the nonsmoker (45).

A number of investigators have found an association between exposure to asbestos and the subsequent development of laryngeal carcinoma (43).

Oral

Oral neoplasms include cancer of the lip, tongue, gums, buccal mucosa, hard and soft palate, salivary glands, floor of the mouth, and oropharynx. In the United States for 1980, there will be 17,900 new cases in men and 7,600 in women, resulting in 6,100 deaths in men and 2,700 deaths in women (1). While different histological types of cancer can occur in this group, squamous cell carcinoma is by far the most common, except for the tumors of the salivary glands. Five-year survival rates range from 25 percent in those patients whose tumor is advanced when first diagnosed to 67 percent for those whose tumor is localized at diagnosis.

In women, oral cancers account for 1.9 percent of all neoplasms, while they account for 4.7 percent of all cancer occurring in men. Deaths from the various oral cancers account for 1.4 percent of cancer deaths in women and 2.8 percent of all cancer deaths in men. Cigarette, pipe and/or cigar smoking are all associated with increased oral cancers. Heavy alcohol use (over 7 ounces per day) has been shown to be an independent causative factor (32,42). When both are used together by women or men, synergism results in an even greater incidence of oral cancer (3). Poor oral hygiene or inadequate dentition is also a risk factor (15).

Most of the prospective epidemiologic studies have concentrated on men. In Japan a large prospective study showed the mortality ratio for oral cancer to be 2.88 for the male cigarette smoker and 1.22 for the female cigarette smoker compared with the nonsmoker.

Leukoplakia or an abnormal thickening and keratinization of the oral mucous membrane is recognized as a precancerous condition. While found in the western world, it is most common in Asian countries where a mixture of tobacco and betel nut or lime ash chewing is common, and in those countries where reverse chutta (cigar) smoking occurs. Women in certain regions of India are more likely to engage in reverse chutta smoking than men, although both women and men develop carcinoma of the hard palate after years of reverse chutta smoking (30).

Women and men with mouth, pharynx, and larynx cancer who continue smoking after surgical treatment of the first neoplasm have a 40 percent probability of developing another neoplasm of the head and neck. Only 6 percent of the patients who quit smoking develop a second cancer in the region. Less than 10 percent of oral cancer patients are nonusers of tobacco; almost all have a well-differentiated carcinoma and a relatively high cure rate (23).

Esophagus

Carcinoma of the esophagus will be diagnosed in 6,200 men and 2,600 women in the United States in 1980 (1). The American Cancer Society estimates that there will be 5,500 deaths in men and 2,100 deaths in women from this disease (1). Median survival time once esophageal carcinoma is diagnosed is 6 months. The 5-year survival rate is only 3 percent. Esophageal carcinoma rates have declined in the white population over the past 25 years. However, they have increased in the black population in both sexes. This may reflect genetic or environmental factors. In the Caspian littoral, there is a remarkable difference in esophageal carcinoma incidence in people of comparable background and socioeconomic status living only 400 kilometers apart. There is a 30-fold higher incidence in women living in the desert northwest section of Mazandran, Iran, compared with the fertile Caspian rainbelt 400 kilometers to the west (20).

Data from a number of retrospective studies show that smoking increases the risk of developing esophageal carcinoma. Neither the relative risk of developing esophageal carcinoma nor the steepness of the dose-response relationship with cigarette smoking is as great as it is for carcinoma of the lung or larynx (45). Individuals who stop smoking or switch to low-tar, low-nicotine cigarettes will, after a lag period, experience lower relative risks of developing esophageal carcinoma, although the fall-off is not as steep as with lung and laryngeal cancer. In the male, both retrospective and prospective studies show that pipe and cigar smokers have mortality rates from esophageal carcinoma similar to cigarette smokers. There are no prospective epidemiologic studies of female smokers in this country large enough to permit development of a mortality ratio comparison to nonsmoking females.

Ingestion of alcohol is also a major etiological factor in esophageal carcinoma. A dose-response relationship exists, with increasing alcohol ingestion resulting in an increased incidence of esophageal carcinoma. As in the larynx, synergism of the carcinogenic effect on the esophagus occurs with the use of both tobacco and alcohol (45). Whether or not nutritional deficiencies, which occur frequently with severe, chronic alcoholism, play a role in carcinogenesis remains unknown, as does the possible contribution of chronic iron deficiency found in Plummer Vinson's syndrome (Paterson-Kelly syndrome, sideropenic dysphagia).

Ninety-eight percent of esophageal cancers are histologically squamous cell in type. In an autopsy study, Auerbach found more abnormalities of the esophageal tissues—including atypical nuclei, disintegrated nuclei, hyperplasia and hyperactive esophageal glands—of tobacco smokers as compared with nonsmokers (2).

Esophageal carcinoma can be produced experimentally by both benz(a)pyrene and the nitrosamines. Both benz(a)pyrene and a group of nitrosamines have been identified in tobacco smoke. The appearance of experimentally-produced squamous cell carcinomas can be accelerated by dissolving the carcinogen in alcohol, a laboratory experiment duplicated daily by thousands if not millions of our citizens (43).

Urinary Bladder

Cancer of the urinary bladder will occur in 26,000 men and 9,500 women in the United States during 1980 and it will kill 7,000 men and 3,300 women (1). Cancer of the urinary bladder is frequently multicentric in origin. If found while still localized in the bladder wall, the 5-year survival rate is 72 percent, in contrast to 14 percent for those patients whose disease had already spread when the diagnosis was first established (1).

Bladder cancer has been associated with occupational exposure to aniline dyes, leading to the study of aromatic amines as potential carcinogens. 2-Naphthylamine, xenylamine, benzidine, and 4-nitrobiphenyl have all been implicated (43).

Numerous retrospective studies have shown a relationship between smoking and urinary bladder carcinoma in both men and women (17). The likelihood of either women or men developing bladder cancer increases with the number of cigarettes smoked, the duration of smoking, and tar and nicotine content of the cigarette smoked. Changing to low-tar, low-nicotine cigarettes or more clearly, cessation of smoking, decreases the relative risk of developing bladder cancer. The risk of an ex-smoker developing urinary bladder cancer approaches that of the nonsmoker years after cessation (46).

In prospective studies in Japan and Sweden, women who smoke are 1.6 to 2.7 times as likely to develop bladder cancer as nonsmokers (3,14). In an international study of successive birth cohorts in the United States, United Kingdom, and Denmark, Hoover and Cole found increasing rates of bladder cancer associated with increased cigarette smoking in men and women in both suburban and rural areas and in all nationalities studied (17). It has been estimated that 30 percent of urinary bladder cancer in women can be attributed to cigarette smoking (43).

Kidney

Cancer of the kidney will occur in 10,500 men and 6,400 women in the United States during 1980 (1). Some 4,800 men and 3,100 women will die of renal carcinoma (1). The 5-year survival rate is between 40 and 50 percent (1). While the overall classification of kidney carcinoma includes tumors of the renal pelvis and ureter, the largest number of kidney carcinomas occur in the renal parenchyma and are adenocarcinomas.

In retrospective studies, adenocarcinomas of the kidney are found more frequently in smokers compared with non-smokers in both men and women (43,44). In a large prospective study among U.S. veterans, the kidney cancer mortality ratio increased from 1.0 (the baseline for nonsmokers) to 1.34 for those who smoked 10 to 19 cigarettes daily and to 2.75 for men who smoked two packs or more each day (18). No large scale prospective study of women and kidney cancer has been reported to date.

Pancreas

Carcinoma of the pancreas will occur in 12,500 men and 11,500 women in the United States during 1980, and 11,100 men and 9,800 women will die of pancreatic carcinoma (1). During the past 25 years, there has been a steady increase in both the incidence and mortality due to pancreatic cancer in both men and women (1,21). Among the common human neoplasms, the rate of increase of pancreatic cancer over the past quarter century has been second only to that of the lung.

Most pancreatic carcinomas are adenocarcinomas, arising from ductal cells (24). Most are relatively undifferentiated in cell type. The median survival time from histologic proof of diagnosis to death is 3.5 months in men and 4.5 months in women. Survival time varies little with age at time of diagnosis, duration of symptoms, location of primary lesion (head, body, or tail of pancreas) or even degree of differentiation. The 5-year survival rate is one percent, the most dismal survival rate for any of the common neoplasms of either men or women (1).

Retrospective studies relating smoking to pancreatic carcinoma have been reviewed in previous reports. In a prospective study of 143,000 women, the pancreatic cancer mortality ratio was 1.94 for Japanese women smokers compared to nonsmokers (14). In Sweden, a smaller prospective study showed that the mortality ratio for pancreatic cancer was 2.5 for women smokers compared to women nonsmokers (4).

In the United States, the male to female ratio of pancreatic cancer was 1.6 in the 1940s. It has decreased to the current estimate of 1.17 for 1979 and is consistent with the decreasing male to female ratios of lung and laryngeal carcinomas.

Summary

1. Cigarette smoking is causally associated with cancer of the lung, larynx, oral cavity, and esophagus in women as well as in men; it is also associated with kidney cancer in women.

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2. Cigarette smoking accounts for 18 percent of all newly diagnosed cancers and 25 percent of all cancer deaths in women. In 1980, 26,500 of the estimated 101,000 deaths, or over one-quarter of the deaths expected from lung cancer, will occur in women.

3. Women cigarette smokers have been reported to have between 2.5 and 5 times greater likelihood of developing lung cancer than nonsmoking women.

4. Among women the risk of developing lung cancer increases with increasing number of cigarettes smoked per day, duration of the smoking habit, depth of inhalation, and tar and nicotine content of the cigarette smoked. The risk is inversely related to the age at which smoking began.

5. A dose-response relationship has been demonstrated between cigarette smoking and cancer of the lung, larynx, oral cavity, and urinary bladder in women.

6. The rise in lung cancer death rates is currently much steeper in women than in men. It is projected that the age adjusted lung cancer death rate will surpass that of breast cancer in the early 1980s.

7. The rapid increase in lung cancer rates in women is similar to but steeper than the rise seen in men approximately 25 years earlier. This probably reflects the fact that women first began to smoke in large numbers 25–30 years after the increase in cigarette smoking among men. Thus, neither men nor women are protected from developing lung cancer caused by cigarette smoking.

8. Cigarette smoking has been causally related to all four of the major histologic types of lung cancer in both women and men, including epidermoid, small cell, large cell and adenocarcinoma.

9. The use of filter cigarettes and cigarettes with lower levels of "tar" and nicotine by women is correlated with a lower risk of cancer of the lung and larynx compared to the use of high-"tar" and nicotine or unfiltered cigarettes. The risk posed by smoking low-"tar" cigarettes, however, is clearly greater than that among females who never smoked.

10. After cessation of cigarette smoking, a woman's risk of developing lung and laryngeal cancer has been shown to drop slowly, equalling that of nonsmokers after 10–15 years.

11. Excessive ingestion of alcohol acts synergistically with cigarette smoking to increase the incidence of oral and laryngeal cancer in women.

References

(1) AMERICAN CANCER SOCIETY. 1979 Cancer Facts and Figures. New York, American Cancer Society, Inc., 1978, 32 pp.

(2) AUERBACH, O., STOUT, A.P., HAMMOND, E.C., GARFINKEL, L. Histologic changes in esophagus in relation to smoking habits. Archives of Environmental Health 11(1): 4–15, July 1965.

(3) BROSS, I.J.D., COOMBS, J. Heavy drinking, smoking, linked with oral cancer. Journal of the American Medical Association 236(5): 435, 1976.

(4) CEDERLOF, R., FRIBERG, L., HRUBEC, Z., LORICH, U. The relationship of smoking and some social covariables to mortality and cancer morbidity. A ten year follow-up in a probability sample of 55,000 Swedish subjects, age 18–69. Part 1 and Part 2. Stockholm, Sweden, The Karolinska Institute, 1975, pp. 1–91.

(5) DOLL, R. The age distribution of cancer. Implications for models of carcinogenesis. Journal of the Royal Statistical Society 134 (Part 2): 133–161, 1971.

(6) DOLL, R., GRAY, R., PETO, R. Mortality in relation to smoking. (Unpublished manuscript)

(7) DOLL, R., PETO, R. Cigarette smoking and bronchial carcinoma dose and time relationships among regular and lifelong nonsmokers. Journal of Epidemiology and Community Health 32: 303–313, 1978.

(8) DOLL, R., PETO, R. Mortality in relation to smoking: 20 years' observations on male British doctors. British Medical Journal 2(6051): 1525–1536, December 25, 1976.

(9) FRAUMENI, J. Genetic Factors in Cancer. In: Holland, J.F., Frei, E. (Editors). Philadelphia, Lea and Febiger, 1973, pp. 7–15.

(10) HAENSZEL, W., TAUBER, K.E. Lung-cancer mortality as related to residence and smoking histories. II. White females. Journal of the National Cancer Institute 324: 803–838, April 1964.

(11) HAMMOND, E.C. Smoking in relation to the death rates of one million men and women. In: Haenszel, W. (Editor). Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases. National Cancer Monograph No. 19. Department of Health, Education and Welfare, Public Health Service, National Cancer Institute, 1966, pp. 127–204.

(12) HAMMOND, E.C., GARFINKEL, L., SEIDMAN, H., LEW, B.A. Some recent findings concerning cigarette smoking in origins of human cancer. In: Hiatt, H.H., Watson, J.D., Winsten, J.A. (Editors). Origins of Human Cancer. Book A: Incidence of Cancer in Humans. New York, Cold Spring Harbor Laboratory, 1977, pp. 101–112.

(13) HARRIS, C.C. (Editor). Pathogenesis and Therapy of Lung Cancer. New York, Dekker, 1978.

(14) HIRAYAMA, T. Prospective studies on cancer epidemiology based on census population in Japan. In: Bucalossi, P., Veronesi, U., Casinelli, M. (Editors). Cancer Epidemiology, Environmental Factors. Volume 3. Proceedings XI International Cancer Congress, Florence, October 20–26, 1974. Amsterdam, Excerpta Medica, 1975, pp. 26–35.

(15) HOLLAND, J.F., FREI, E. (Editors). Cancer Medicine. Philadelphia, Lea and Febiger, 1973.

(16) HOOVER, R., COLE, P. Population trends in cigarette smoking and bladder cancer. American Journal of Epidemiology 94(5): 409–418, 1971.

(17) HORN, D. The benefits of stopping smoking. In: Steinfeld, J., Griffiths, W., Ball, K., Taylor, R.M. (Editors). Proceedings of the Third World Conference on Smoking and Health. Volume II. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute, DHEW Publication No. (NIH) 77-1413, 1977, pp. 59–64.

(18) KAHN, H.A. The Dorn study of smoking and mortality among U.S. veterans. Report on eight and one-half years of observations. In: Haenszel, W. (Editor). Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases. National Cancer Institute Monograph No. 19. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute, January 1966, pp. 1–27, 124–125.

(19) KEMP, I.W., RUTHVEN, H.E. Cancer of the lungs in Scotland. Health Bulletin, pp. 259–268, 1979.

(20) KMET, J.A. AND MAHBOUBI, E. Esophageal cancer in the Caspian littoral of Iran: initial studies. Science 175: 846, February 25, 1972.

(21) KRAIN, L.S. The rising incidence of carcinoma of the pancreas, real or apparent? Journal of Surgical Oncology 2(2): 115–124, 1970.

(22) KREYBERG, L. Histologic typing of lung tumors. International Histological Classification of Tumors No. 1. Geneva, Switzerland, World Health Organization, 1967.

(23) MOORE, C. Smoking related to cancer of the mouth, tongue and lip. In: Steinfield, J., Griffiths, W., Ball, K., Taylor, R.M. (Editors). Proceedings of the Third World Conference on Smoking and Health, New York, June 2–5, 1975. Volume II. Health Consequences, Education, Cessation Activities, and Social Action. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute. DHEW Publication No. (NIH) 77-1413, 1977, pp. 101–104.

(24) MORTEL, C. In: Holland, J., Frei, E. (Editors). Cancer Medicine. Philadelphia, Lea and Febiger, 1973.

(25) NATIONAL CANCER INSTITUTE. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute, Surveillance, Epidemiology, and End Results (SEER) Program. (Unpublished data)

(26) NATIONAL CANCER INSTITUTE. Carcinogenesis Technical Report Series, 1977–1979. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute.

(27) NATIONAL CENTER FOR HEALTH STATISTICS. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics. (Unpublished data)

(28) NATIONAL CENTER FOR HEALTH STATISTICS. Health, United States, 1978. Department of Health, Education, and Welfare, Public Health Service, Office of Health Policy, Research and Statistics, National Center for Health Statistics, DHEW Publication (PHS) 78-1232, 1979.

(29) NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH. Adult Use of Tobacco, 1975. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, Bureau of Health Education, National Clearinghouse for Smoking and Health, June 1976, 23 pp.

(30) REDDY, C.R.R.M., SEKHAR, C., RAJU, M.V.S., REDDY, S.S., KAMESWARI, V.R. Relation of reverse smoking to carcinoma of the hard palate. Indian Journal of Cancer 8(4): 262–268, December 1970.

(31) ROSENOW, E.C., CARR, D.T. Bronchogenic carcinoma. CA 29(4): 233–245, 1979.

(32) ROTHMAN, K., KELLER, A. The effect of joint exposure to alcohol and tobacco on risk of cancer of the mouth and pharynx. Journal of Chronic Disease 25: 711–716, 1972.

(33) U.S. PUBLIC HEALTH SERVICE. Smoking and Health. Report of the Advisory Committee to the Surgeon General of the Public Health Service, Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, DHEW Publication No. 1103, 1964, 387 pp.

(34) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking. A Public Health Service Review: 1967. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration, DHEW Publication No. 1696, January 1968, 227 pp.

(35) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking, 1968. Supplement to the 1967 Public Health Service Review. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration, Publication No. 1696, 1968, 117 pp.

(36) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking, 1969. Supplement to the 1967 Public Health Service Review. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration, DHEW Publication No. 1969–2, 1969, 98 pp.

(37) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration, DHEW Publication No. (HSM) 71-7513, 1971, 458 pp.

(38) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration, No. (HSM) 72-7516, 1972, 158 pp.

(39) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking, 1973. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration, DHEW Publication No. (HSM) 73-8704, 1973, 249 pp.

(40) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking, 1974. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, DHEW No. (CDC) 74-8704, 1974, 124 pp.

(41) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking, 1975. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, DHEW Publication No. (CDC) 76-8704, 1976, 235 pp.

(42) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking. A Reference Edition. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, DHEW Publication No. (CDC) 78-8357, 1976, 657 pp.

(43) U.S. PUBLIC HEALTH SERVICE. Smoking and Health. A Report of the Surgeon General. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, Office of Smoking and Health, DHEW Publication No. (PHS) 79-50066, 1979, 1251 pp.

(44) WYNDER, E.L., MABUCHI, K., WHITMORE, F.W., JR. Epidemiology of adenocarcinoma of the kidney. Journal of the National Cancer Institute 53(6): 1619–1634, December 1974.

(45) WYNDER, E.L., STELLMAN, S.D. Comparative epidemiology of tobacco-related cancers. Cancer Research 37: 4608–4622, December 1977.

(46) WYNDER, E.L., STELLMAN, S.D. The impact of long-term filter cigarette usage on lung and larynx cancer. Journal of the National Cancer Institute 62(13): 471–477, March 1979.

NON-NEOPLASTIC BRONCHOPULMONARY DISEASES

Introduction

Chronic non-neoplastic bronchopulmonary disorders are a major cause of death and disability in the United States. Chronic obstructive lung diseases (COLD), including chronic bronchitis and emphysema, comprise the majority of these illnesses. In 1977, they were responsible for nearly 46,000 deaths and millions of dollars in social security disability payments, ranking second in economic cost only to heart disease (42).

Previous U.S. Public Health Service reports on the health consequences of smoking have presented evidence that cigarette smoking is the major cause of COLD (55–64). The studies on which this is based have focused primarily on male populations. This reflects the scientific interest generated by the overwhelming male-to-female ratio in the prevalence of COLD at the time these studies began. However, recent mortality statistics indicate a substantial increase in the death rate from COLD among women (see Mortality section). Although this increased death rate may partially reflect a greater awareness and recognition of COLD, its magnitude suggests a true increase in frequency of COLD among women. The following text reviews a large number of studies analyzing the relationship of smoking to COLD. These studies include appreciable numbers of women, and many suggest that smoking may affect men and women differently. Nevertheless, cigarette smoking remains the most important cause of COLD regardless of sex or other variables.

Definitions

The terms chronic bronchitis and emphysema have been used diagnostically for many years. Physicians often use these terms interchangeably to describe a patient with chronic airflow obstruction. These conditions are, however, difficult to distinguish from each other in patients with chronic airflow obstruction because (a) both conditions may be present in the same patient; (b) both disorders are characterized by expiratory flow obstruction; and (c) patients with either disorder frequently have the same symptom—dyspnea on exertion. Consequently, the clinician often labels the patient with chronic airflow obstruction as having chronic obstructive lung disease (COLD). Many attempts have been made to establish criteria for the diagnosis of chronic bronchitis and emphysema (1,27,28). The most widely accepted definitions in the United States are those

TABLE 1.—Age-adjusted death rates from COLD (ICDA 490-492 and 519.3) 1960-1977 (per 100,000)

	White		Nonwhite	
	Male	Female	Male	Female
1977	33.4	10.7	14.8	3.5
1976	33.5	10.1	14.9	3.2
1975	32.1	9.1	13.5	3.3
1974	31.1	8.4	13.7	2.8
1973	31.4	7.8	14.1	3.0
1972	29.9	7.0	14.0	2.9
1971	28.6	6.5	13.2	3.0
1970	28.2	6.0	13.3	2.6
1969	27.3	5.4	12.8	2.4
1968	22.3	3.8	13.7	2.5
1967	19.9	3.1	11.5	2.0
1966	19.7	3.0	11.0	1.9
1965	18.4	2.7	10.4	1.8
1964	16.1	2.4	9.2	1.6
1963	15.9	2.3	9.5	1.9
1962	13.1	2.0	7.7	1.8
1961	10.9	1.7	7.0	1.3
1960	10.4	1.7	6.7	1.4

SOURCE: National Center for Health Statistics (42).

of a joint committee of the American College of Chest Physicians and the American Thoracic Society (1).

"Bronchitis: A non-neoplastic disorder of structure or function of the bronchi resulting from infectious or noninfectious irritation. The term bronchitis should be modified by appropriate words or phrases to indicate its etiology, its chronicity, the presence of associated airways dysfunction or type of anatomic change. The term chronic bronchitis, when unqualified, refers to a condition associated with prolonged exposure to nonspecific bronchial irritants and accompanied by mucous hypersecretion and certain structural alterations in the bronchi. Anatomic changes may include hypertrophy of the mucous-secreting apparatus and epithelial-metaplasia, as well as more classic evidence of inflammation. In epidemiologic studies, the presence of cough or sputum production on most days for at least 3 months of the year has sometimes been accepted as a criterion for diagnosis."

"Pulmonary Emphysema: An abnormal enlargement of the air spaces distal to the terminal nonrespiratory bronchiole, accompanied by destructive changes of the alveolar walls. The term emphysema may be modified by words or phrases to indicate its etiology, its anatomic subtype, or any associated airway dysfunction."

"Chronic Obstructive Lung Disease: This term refers to a disease of uncertain etiology characterized by persistent slowing of airflow during forced expiration. It is recommended that a more specific term, such as chronic obstructive bronchitis or chronic obstructive emphysema, be used whenever possible."

It should be noted that these definitions may have serious inadequacies, particularly when applied to longitudinal studies assessing the natural history of COLD (29,52). In the following discussion, these limitations are recognized.

Smoking and Respiratory Mortality

Recent mortality statistics indicate a striking increase in death rate from COLD among women (42). These data presented in Table 1 indicate a nearly fivefold increase in reported mortalities due to COLD from 1962 to 1977 among white females and a twofold increase among nonwhite females. Mortality rates from these conditions for white and nonwhite males have also increased since 1967 (by factors of 1.9 and 1.5, respectively), but the rate of increase has not been as steep as that for women.

Seven large prospective studies have shown a greatly increased mortality from COLD among smokers as compared to nonsmokers (14,18,19,31,32,37). These studies, presented in Table 2, represent over 13 million subject years of observation and approximately 270,000 deaths from all causes. The number of deaths related to COLD is probably underestimated since some of the deaths attributed to pneumonia or myocardial disease may have been due to complications of COLD. In addition, these mortality figures do not include an appreciable number of individuals for whom COLD may have been a major contributory cause of death. For example, it is not uncommon for individuals to have COLD and lung cancer simultaneously.

Two of these prospective studies have included significant numbers of women. Hammond prospectively followed 1,003,229 subjects aged 35 to 84 (31). Nearly 93 percent of the survivors were observed for a 12-year period. Death rates from emphysema among women were much higher in cigarette smokers than nonsmokers. "Heavier" smokers (defined as either smokers of 20 or more cigarettes a day regardless of age when smoking was begun, or smokers of 10 or more cigarettes a day who had begun smoking before age 25) had a sevenfold increased mortality rate as compared to nonsmokers. Cederlof et al. followed 55,000 Swedish subjects aged 10 to 69 for 10 years (14). The overall mortality rate from all causes among female smokers was 1.2 times higher than that of female nonsmokers. The death rate from bronchitis, emphysema, and asthma among

TABLE 2.—COLD mortality ratios + in seven prospective studies

Study (Reference)	British Doctors (18)	Women in 25 States 45–65 (31)	Men in 25 States (31) 45–64	65–79	U.S. Veterans (37)	Canadian Veterans (8)	Men in 9 States (32)	California Occupations (19)	Swedish Subjects (14) Females	Males
Emphysema and/or bronchitis	24.7	—	—	—	10.08	—	2.30	4.3	—	—
Emphysema without bronchitis	—	4.89	6.55	11.41	14.17	7.7	—	—	—	—
Bronchitis	—	—	—	—	4.49	11.3	—	—	—	—
Bronchitis, emphysema and asthma	—	—	—	—	—	—	—	—	2.2	3.7*

+ Death rate for smokers divided by death rate of a comparable group of nonsmokers.

*For all ages combined; increased mortality rate significant only for former smokers.

female smokers was 2.2 times that of female nonsmokers. However, the number of deaths due to COLD among women was small in both of these studies; consequently, the relationship with smoking is more difficult to evaluate. Nevertheless, a significant excess risk for reported mortality from COLD was present for female cigarette smokers as compared to female nonsmokers.

Data collected by Doll et al. examine the association of smoking and cause-specific mortality in 6,194 women physicians in England, observed prospectively over the period 1951 to 1973 (17). Table 3 presents the results of this study, including previously published results of a similar study among male physicians over the same period (18). The association of smoking and chronic bronchitis clearly observed in males was confirmed in women physicians. For both women and men who reported smoking 15 or more cigarettes per day, the mortality rate due to emphysema and chronic bronchitis was more than five times as great as in nonsmokers. In both sexes, mortality due to emphysema and chronic bronchitis was more than double that of nonsmokers, was at least three times as high in ever-smokers as in never-smokers, and was at least twice as high in current heavy smokers (≥25 cigarettes) as in light smokers (≤15 cigarettes).

The risk of death from emphysema and chronic bronchitis associated with smoking was approximately similar in men and women. For moderate (1 to 14 cigarettes per day) and heavy (≥25 cigarettes per day) smokers, compared with nonsmokers, the relative risk of death was 28.5 and 32 for women, respectively, versus 16.7 and 29.3 for men. In this data, as well as that for lung cancer, there is no support for the contention that women are less susceptible to harmful effects of smoking than are men. The authors emphasize that no conclusions can be drawn from this data about the magnitude of the biologic effects of smoking in men compared to women. Attempts to document differences in lifetime smoke exposure (later age at initiation and lower prevalence of inhalation among females) demonstrate that lifetime smoking exposures between the sexes are not comparable. This issue will be resolved only when studies examine the effect of smoking in cohorts of women whose lifetime smoking behavior more closely matches that of the men to whom they are compared.

In comparing the relative risks for mortality from COLD in female and male smokers (Table 2), it is apparent that female smokers have lower reported mortality rates than their male counterparts. This difference in mortality rates may be due to differences in female smoking patterns (31). Women tend to

TABLE 3.—Death rates from chronic bronchitis and emphysema by smoking habit when last asked, British physicians 1951-1973

			Annual Death Rate Per 100,000 Persons Standardized for Age					X^2	
					Current Smokers—Dose Per Day				
	Total Popul.	# Deaths	Non-Smokers	Ex-Smokers	1-14	15-25	>25	Nonsmokers vs. All Others	Trend (Dose/Response)
Women	6,194	13	2	10	21	57 (cigarettes only)	64	12.34*	26.64*
Men	34,440	254	3	44	38	50 (any tobacco/grams) (1 gram = 1 cigarette)	88	25.58*	47.23*

*(P >0.001)

SOURCE: Doll, R. (17,18).

smoke fewer cigarettes, inhale less deeply, and begin smoking later in life than men. They more frequently smoke filtered and low-tar and -nicotine cigarettes and have less occupational exposure to lung irritants than men. Recent data suggest that women are manifesting smoking patterns similar to those of men. Moreover, more women are joining the labor force, including occupations where exposure to lung irritants may occur. (See section on Occupational Exposures.) Whether these women will continue to have mortality rates different from those of men remains to be determined.

In summary, recent statistics indicate a rise in the reported death rate due to COLD among women. The two large prospective studies that included appreciable numbers of women found significantly higher mortality rates due to COLD among women smokers as compared to women nonsmokers. This relationship was accentuated in heavier smokers. Mortality rates from COLD among female smokers are considerably lower than among male smokers. This may be due to different smoking patterns and work exposure among men and women.

Smoking and the Epidemiology and Pathology of COLD

The prevalence of chronic bronchitis has been determined in several populations in the United States and in other countries (24,25,26,34,36,41,43,44,46,51). Table 4 lists several studies which have included appreciable numbers of women. These studies have documented a close relationship between cigarette smoking and an increased prevalence of chronic bronchitis, and when looked for, a dose-response relationship was also present (Table 3). The prevalence of chronic bronchitis in the United States was determined in four cohort studies and ranged from 4 to 10 percent among women and 14 to 18 percent among men (24,25,26,41,44,51). In both men and women a dose-response relationship between the number of cigarettes smoked and the prevalence of chronic bronchitis was apparent.

The observed differences between men and women noted in these studies may be due in part to the smaller percentage of women than men who were smokers in the population studied. Moreover these women smoked fewer cigarettes than men. When comparing current smokers, several studies of different populations in the United States and in England did not find significant differences in the prevalence of chronic bronchitis between men and women (21,33,41).

The relationship between smoking and pathologic changes in the lung have largely been obtained by necropsy studies. These investigations are often skewed by physician and/or hospital

TABLE 4.—Prevalence of chronic bronchitis by smoking classification (numbers in parentheses represent total number of individuals in particular smoking group)

S = Smokers NS = Nonsmokers EX = Ex-Smokers

Author, Year Country (Reference)	Number and Type of Population	Men	Women	Comment
Higgins, 1958 England (34)	94 men and 92 women randomly chosen from agricultural communities	NS .... 0.0 S 6.7	NS .... 0.0 S 5.0	
Oswald, 1955 England (43)	3,602 males and 2,242 female clerical workers 40-65 yrs. of age	NS .... 15.8 (474) S 18.4 (1,940)	NS .... 12.1 (619) S 18.8 (579)	Chronic bronchitis defined by habitual cough and sputum production
Hubti, 1965 England (36)	653 men and 823 women in a Finnish rural community 40-60 yrs. of age	NS .... 5.7 EX .... 16.3 S 1-14 .... 38.0 15-24 .... 41.4 >25 .... 4.0	NS .... 4.5 EX .... 13.3 S 1-14 .... 10.4 15-24 .... >25 .... 57.0	Ex-smokers represent those who have stopped for more than 1 month
Remington, 1969 England (46)	41,729 men and 22,295 women participating in mass miniature radiography screening	NS .... 5.1 (9,055) EX .... 9.8 (6,510) Cigarettes .... (23,243) S 1-19 .... 9.1 10-19 .... 15.0 >20 .... 20.6	NS .... 3.4 (12,351) EX .... 3.9 (959) Cigarettes .... (8,985) S 1-9 .... 5.1 10-19 .... 10.6 >20 .... 18.5	Age-adjusted total prevalence. Cigarette dosage gradient significant to $P < 0.001$

Ferris, 1962 U.S.A. (23,25,26)	542 men and 625 women residents of New Hampshire town chosen by random sampling of census	Overall NS .... 13.8 (125) EX .... 11.9 (77) Cigarettes ... 40.3 (340) 1-10 .... 29.8 11-20 .... 34.2 21-30 .... 42.3 31-40 .... 61.1 >41 .... 75.3	Overall NS .... 9.4 (378) EX .... 10.8 (37) Cigarettes ... 19.8 (208) 1-10 .... 13.1 11-20 .... 22.2 21-30 .... — 31-40 .... 27.3 >41 .... —	Age-specific rates
Payne, 1964 U.S.A. (44)	5,140 adult residents of Tecumseh, Mich.	Overall 8	Overall 4	Prevalence rates estimated from line graph
Mueller, 1971 U.S.A. (41)	281 men and 328 women residents of Glenwood Springs, Colo.	Overall .... 17 (281) NS .... 3 (2) EX .... 13 (7) S 1-14 .... 11 (3) 15-24 .... 20 (13) >25 .... 38 (21)	Overall .... 10 (328) NS .... 2 (3) EX .... 5 (1) S 1-14 .... 14 (7) 15-24 .... 25 (14) >25 .... 33 (9)	
Tager, 1976 U.S.A. (51)	227 men and 280 women in East Boston, Mass. age 15 or greater	Overall ...... 14.7 (227) NS .... 5.8 S 24.2	Overall ...... 7.5 (285) NS .... 1.8 S 17.6	Age-adjusted prevalence rate

interest and may not accurately represent a random population. Moreover, observer variation occurs frequently, even among "experts." Data regarding smoking history are usually derived from a hospital record or from close relatives and friends; thus they may be unreliable.

Only a few of the studies examining the relationship of cigarette smoking to the frequency and severity of pathological changes have included significant numbers of female subjects. Thurlbeck recently reviewed 30 reported surveys of the frequency of emphysema at necropsy (53). Emphysema of some degree was found in about 65 percent of men and 15 percent of women. The emphysema found was also more severe in men than in women.

The predominant pathological finding in chronic bronchitis is the hypertrophied mucous gland in the submucosa of the large cartilaginous bronchi. The ratio of bronchial gland thickness to bronchial wall thickness (Reid index) is usually increased. In a recent survey of 179 consecutive necropsies, Ryder et al. found significantly greater bronchial mucous gland volume in smokers compared to nonsmokers. There was no significant difference in mucous gland volume between male and female smokers or male and female nonsmokers (48).

Mueller et al. examined the prevalence of chronic bronchitis in one-fifth of the adult population of Glenwood Springs, Colorado (41). Among current smokers of varying smoking categories (Table 4) there were no significant differences in the prevalence of chronic bronchitis. Higgins and Cochran found no significant difference in the prevalence of chronic bronchitis between men and women smokers in 186 subjects randomly chosen from an agricultural community (Table 4) (34). Similarly, Oswald and Medvel found no significant difference in the prevalence of chronic bronchitis between men and women smokers in 5,844 clerical workers in England (Table 4) (43).

Auerbach et al. examined the relationship of smoking to emphysema in whole-lung and microscopic sections at necropsy in 1,436 men and 388 women (4,5). Among the women, there were 97 current smokers, 16 of whom smoked two packs a day or more. Data regarding smoking habits were obtained through interviews with relatives. Female smokers had a significantly higher rate of emphysema than female nonsmokers (Table 5). Furthermore, the severity of the emphysema was dose-related to the number of cigarettes smoked. The authors found similar relationships in men.

Spain et al. examined consecutive whole-lung mounts from necropsies of adult victims (49 women, 85 men) of sudden and unexpected death (50). Smoking habits were ascertained by a letter and questionnaire to the next of kin. The degree of emphysema was graded from 0 to 100 by two observers independently and without prior knowledge of the source of the specimen or any previous grading. There was a close relationship between cigarette smoking and the degree of emphysema in both men and women. Furthermore, the data (Table 6) demonstrated a dose-response effect between the number of cigarettes smoked and the severity of pathological changes.

Thurlbeck et al. examined whole-lung sections in 1,742 random necropsies in three different cities in different countries with varying climates and environments (54). Using a standard panel of grading pictures, pathologic changes in the lung were graded from 0 to 100 by the three readers. In men and women emphysema was more frequent and more severe in smokers than nonsmokers; however, male smokers had higher average emphysema scores and greater frequency of emphysema than female smokers and nonsmokers. This difference between men and women was also true when heavy smokers and ex-smokers of both sexes were compared. The authors speculate that male-female differences may exist because: (a) women are protected by hormonal factors; (b) men may smoke more heavily than women; (c) men may have different smoking patterns than women, e.g., inhalation; and (d) men may be exposed to damaging environmental factors at work.

TABLE 5.—**Means of average degrees of findings* in nonsmokers and current smokers standardized for age of total study population, women**

	Subjects Who Never Smoked Regularly	Current Cigarette Smokers <1 Pk.	1+Pk.
Number of subjects	252	33	64
Emphysema	0.05	1.37	1.70
Fibrosis	0.37	2.89	3.46
Thickening of arterioles	0.06	1.26	1.57
Thickening of arteries	0.01	0.40	0.64

*The pathologic findings recorded were: (1) degree of emphysema (four-point scale ranging from zero for normal to four for advanced emphysema); (2) degree of fibrosis (seven-point scale ranging from none to advanced diffuse fibrosis); (3) degree of thickening of arterioles (four-point scale); (4) degree of thickening of arteries (three-point scale); and (5) padlike attachments to alveolar septa. Padlike attachment is a thickening of alveolar septa in focal areas by fibroblasts, histocytes and collagen fibrils. This is recorded as present or absent.

SOURCE: Auerbach, O. (4).

In summary, the prevalence of chronic bronchitis among women in the United States has been reported to range from 4 to 10 percent. Women who smoke have a higher prevalence of chronic bronchitis than those who do not smoke. Overall, however, chronic bronchitis is less common among women than men in the United States. This may reflect the smaller proportion of women who smoke, differences in their smoking behavior, and less occupational exposure to lung irritants. When comparing current smokers, several studies of different populations in the United States and England did not find significant differences in the prevalence of chronic bronchitis between men and women. Pathological data suggest that female smokers have a higher frequency of emphysema and bronchial mucous gland hypertrophy than female nonsmokers. Furthermore, the severity of emphysema is dose-related to the number of cigarettes smoked. Distinct female-male differences in the frequency and extent of emphysema at autopsy have been reported, but it is not clear whether these differences are due to intrinsic differences in the way men and women respond to environmental injury or to the differences in the degree of environmental injury experienced by men and women.

Smoking and Respiratory Morbidity

A large number of recent studies have demonstrated a higher frequency of respiratory symptoms, i.e., cough, sputum, wheezing and dyspnea, in smokers as compared to nonsmokers. Many

TABLE 6.—Degree of emphysema* and cigarette smoking**

Cigarettes Per Day	No. Over Age 30	Mean Grade of Emphysema	No. With Grade 20 Emphysema	Mean Age With Grade 20 Emphysema
Men				
0	30	8 (0–20)	3 (10%)	66
<21	14	11 (0–45)	5 (36%)	62
>20	41	14 (0–50)	16 (39%)	52
Women				
0	21	2 (0–10)	0	—
<21	6	6 (0–20)	1 (17%)	70 +
>20	22	8 (0–30)	5 (23%)	40

*x^2 test shows significance at the 1% level for the heavy smokers and nonsmokers.

**Each whole lung paper mounted section was graded from 0 to 100 in denominations of 5 up to grade 50 and then in denominations of 10 up to grade 100.

+One case.

SOURCE: Spain, D.M. (50).

of these studies have included appreciable numbers of women (9,11,15,38,39,40,45,47,65). These investigations have examined populations varying in age, geographic location, social class, and exposure to air pollution.

Leibowitz and Burrows examined the quantitative relationships between cigarette smoking and chronic productive cough in a large randomized sample of the white non-Mexican American population of Tucson, Arizona (38). Their data (Table 7) confirm the close relationship between cigarette smoking and chronic cough and/or chronic sputum production in men and women. The effect of cigarette smoking was closely related to the total pack-years smoked (Table 7). These data support the male to female preponderance in prevalence of chronic bronchitis noted in several other epidemiologic surveys (24,25,26,41,44,51). However, these data also indicate that males and females with equivalent smoking histories have similar rates of chronic cough and/or sputum production.

Woolf examined the frequency of respiratory symptoms in women volunteers, aged 25 to 54, drawn from several large commercial firms (Table 8) (65,66). The prevalence of cough and sputum production was significantly greater in smokers than in nonsmokers ($p < 0.001$). Heavier smokers complained of cough and/or sputum production more frequently than nonsmokers or ex-smokers. The prevalence of wheezing and exertional dyspnea increased progressively with the number of cigarettes smoked. In addition, colds that "went to the chest" occurred more frequently in moderate and heavy smokers than in nonsmokers ($p < 0.005$ and $p < 0.001$, respectively). Woolf compared his data with previously reported data among men (Table 9) and concluded that the relationship of cigarette smoking to respiratory symptoms was similar among men and women.

Ferris resurveyed a 1967 sample of Berlin, New Hampshire, residents in 1973 (22). As in 1967, the prevalence of cough and/or sputum production in females and males was directly related to the number of cigarettes smoked daily. When the group evaluated in 1967 was examined by current inhaling and smoking status (Figure 1), inhalers had a higher prevalence of symptoms than noninhalers (22). Furthermore, the frequency of symptoms was dose-related to the number of cigarettes smoked. Manfreda et al. studied population samples in an urban and a rural community in Manitoba, Canada (39). Their data presented in Table 10 demonstrate a higher prevalence of cough, phlegm, and wheezing among men and women who smoked than in nonsmokers or ex-smokers. However, no significant differences in the prevalence of symptoms were apparent in the two communities.

TABLE 7.—Comparison of prevalence of chronic cough + and/or chronic sputum production + in men and women, by smoking habits*

	(Number of Subjects) % With Symptoms							
	Never Smoked		Ex-Smokers		Presently 1-20/day		Presently >20/day	
A. By age group	Males	Females	Males	Females	Males	Females	Males	Females
15-29 years	(156) 7.2	(182) 8.2	(36) 8.3	(45) 17.7	(78) 25.7	(82) 20.8	(34) 41.2	(17) 41.1
30-44 years	(43) 2.3	(82) 12.2	(45) 11.1	(41) 4.8	(43) 39.5	(40) 35.0	(40) 47.5	(30) 56.7
45-59 years	(45) 11.1	(119) 10.9	(61) 21.3	(63) 20.6	(57) 43.8	(83) 36.2	(54) 61.1	(39) 51.3
60+ years	(105) 18.1	(336) 14.6	(186) 36.0	(77) 20.8	(62) 51.6	(82) 34.1	(16) 81.3	(14) 57.1

B. By pack-years of smoking	Present Smokers		Ex-Smokers	
Never smoked	(350) 10.3	(719) 12.1	(350) 10.3	(719) 12.1
Smoked < 6 pack-years	(69) 29.0	(81) 21.0	(59) 5.3	(69) 15.9
6-20 pack-years	(106) 35.8	(127) 33.1	(77) 14.3	(69) 15.9
21-40 pack-years	(96) 47.9	(126) 40.5	(86) 34.9	(27) 18.5
40+ pack-years	(113) 61.1	(53) 60.4	(106) 35.8	(30) 16.7

*Subjects with a history of childhood respiratory problems have been excluded from the analysis. Differences in rates by smoking significant within each age-sex group (X^2 and z differences between proportions) and trend with smoking significant within age-sex groups (X^2 trend). Trend of symptoms by pack-years significant for male present and ex-smokers and female present smokers (X^2 trend). Never smokers always significantly different from present or ex-smokers (X^2 and z).

+Symptoms are those reported on a self-completion questionnaire and are derived from the National Heart and Lung Institute modification of the British Medical Research Council respiratory questions. "Chronicity" of cough or sputum production refers to the presence of the symptom "on most days for at least three months of the year."

SOURCE: Leibowitz, M. (38).

TABLE 8.—Prevalence of cough and sputum production in 500 women related to smoking habit

	Nonsmokers		Ex-smokers		Light Smokers		Moderate Smokers		Heavy Smokers	
	No.	%	No.	%	No.	%	No.	%	No.	%
a. Cough*	11	6.0	1	1.6	11	27.5	32	34.8	66	53.7
b. Sputum**	14	7.7	1	1.6	12	30.0	27	29.3	60	48.8
c. Sputum volume										
None	169	92.3	61	98.4	28	70.0	65	70.7	63	31.2
Morning blob	10	5.5	0	0.0	7	17.5	11	12.0	29	23.6
Tablespoonful	3	1.6	0	0.0	5	12.5	12	13.0	17	13.8
More than one tablespoonful	1	0.5	0	0.0	0	0.0	4	4.4	12	9.8

*Includes women with cough with or without sputum.

**Includes women with sputum with or without cough.

SOURCE: Woolf, C.R. (65).

TABLE 9.—Prevalence of respiratory symptoms in men compared with women*

	Men (Published Data)	Women (Present Investigation)
Cough	Percent	Percent
Nonsmokers	4 (46) 14-22 (47)	6
Light smokers	24 (48)	28
Moderate smokers	48-52 (48)	35
Heavy smokers	42 (46) 67-74 (47) 58-78 (48)	54
Sputum		
Heavy smokers	42 (46)	49
Dyspnea		
All smokers	21 (49)	27
Heavy smokers	33 (50)	33

*Numbers in parentheses are reference numbers.
SOURCE: Woolf, C.R. (65).

The relationship between smoking and several respiratory symptoms was examined by Buist et al. in population samples of three North American cities (11). Cough, sputum production, and wheezing occurred more frequently among smokers than nonsmokers regardless of sex.

Bewley and Bland examined the relationships between smoking and the prevalence of respiratory symptoms in 14,033 children aged 10 to 12½ in two separate urban areas of the United Kingdom (9). In this questionnaire survey, 2.5 percent of the girls acknowledged smoking at least one cigarette per week ("smoker"). Boys who smoked outnumbered girls who smoked by 3:1 and were more frequent smokers of at least one cigarette a day than were females by 11:1. Table 11 shows that, even in this young age group, smokers have a higher frequency of morning cough, cough during the day and night, and cough for 3-months duration than their nonsmoking classmates.

In a questionnaire study of a large group of American high school students in Rochester, New York, Rush found a strong association between current smoking and respiratory symptoms in both sexes (47). There were minor differences between sexes in the frequency of respiratory symptoms when

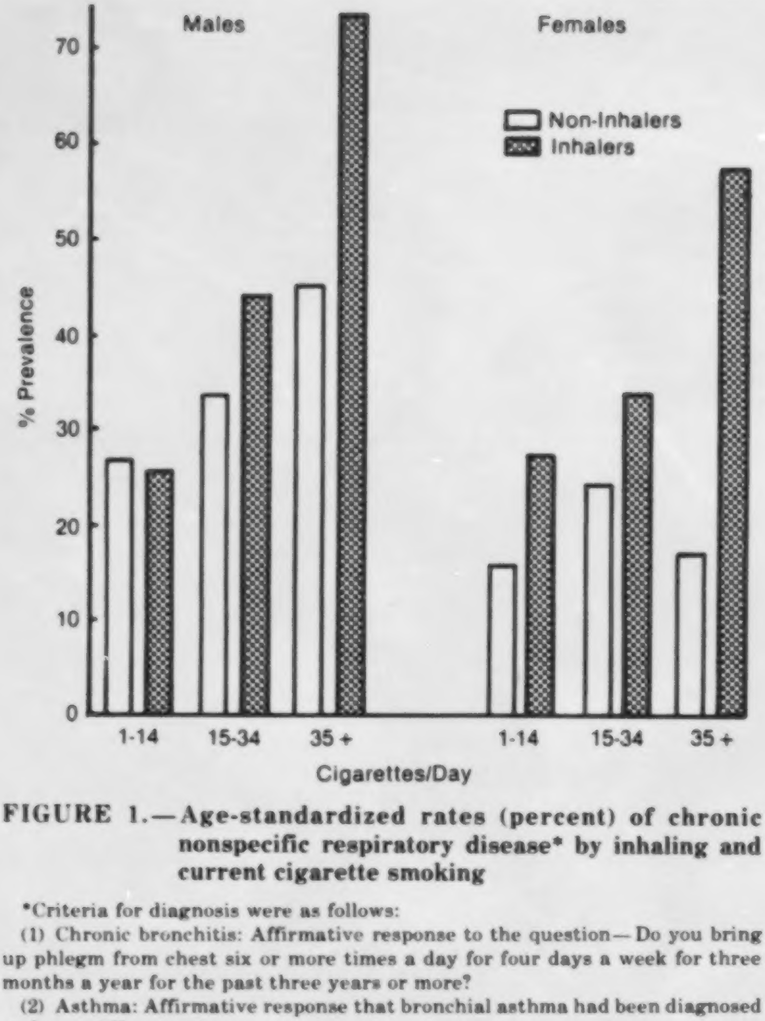

FIGURE 1.—Age-standardized rates (percent) of chronic nonspecific respiratory disease* by inhaling and current cigarette smoking

*Criteria for diagnosis were as follows:

(1) Chronic bronchitis: Affirmative response to the question—Do you bring up phlegm from chest six or more times a day for four days a week for three months a year for the past three years or more?

(2) Asthma: Affirmative response that bronchial asthma had been diagnosed and was still present.

(3) Chronic obstructive lung disease: Affirmative response to one or more of the following: wheezing or whistling in the chest occurred most days or nights; the subject had to stop for breath when walking at his own pace on the level; FEV_1 less than 60 per cent of the FVC.

These could occur in various combinations and were not mutually exclusive.

SOURCE: Ferris, B.G., Jr. (22).

smoking histories were comparable. Rawbone et al., in a questionnaire survey of 10,498 secondary school children aged 11 to 17 in London, found a significantly higher frequency of cough,

TABLE 10.—Respiratory symptoms and diseases in male (M) and female (F) participants in Charleswood (C)—urban—and in Portage La Prairie (P)—rural—expressed as percent of respondents

Respiratory Symptom/Disease	Nonsmokers C	Nonsmokers P	Ex-Smokers C	Ex-Smokers P	Smokers C	Smokers P
Cough on most days, at least 3 months/year						
M	8.3	4.0	8.1	2.9	25.4	31.5
F	—	4.0	—	10.0	20.3	31.7
Phlegm on most days, at least 3 months/year						
M	—	4.0	10.8	5.7	16.9	24.7
F	—	4.0	—	5.0	10.2	25.4
Wheezing apart from colds						
M	4.2	8.0	10.8	14.3	26.8	31.5
F	3.5	8.0	12.1	20.0	25.4	30.2
Attack of shortness of breath and wheezing						
M	4.2	8.0	13.5	11.4	11.3	17.8
F	—	12.0	6.1	15.0	13.5	20.6
Shortness of breath compared to persons of same sex and age						
M	8.3	4.0	5.4	5.8	5.6	12.3
F	7.0	12.0	6.1	5.0	22.1	17.5

SOURCE: Manfreda, J. (39).

colds, and exertional dyspnea in regular smokers as compared to nonsmokers (45). There was no appreciable difference in the frequency of cough between male and female smokers or between male and female nonsmokers. Colley et al. examined the influence of smoking, lower respiratory tract illness under 2 years of age, social class of father, and air pollution on respiratory symptoms in a cohort of 20-year-olds followed since birth (15). Their data (Table 12) suggest that respiratory symptoms were closely related to current smoking. Symptoms were also related to a history of lower respiratory tract infection in the first 2 years of life but were not related to social class or air pollution.

TABLE 11.—Smoking and the prevalence of respiratory symptoms in girls from two different cities in England

Symptom	Residence	Prevalence of Symptom With Each Group Smoker⁺ N	%	Experimental Smoker† N	%	Nonsmoker N	%	Significance*
Cough in the morning	Kent	10	31.3	51	9.8	73	6.9	P <0.001
	Derbyshire	14	18.9	50	8.4	138	6.7	P <0.001
Cough day or night	Kent	17	53.1	148	28.0	195	18.4	P <0.001
	Derbyshire	35	47.3	176	29.5	458	22.1	P <0.001
Cough for 3 months of year	Kent	5	15.6	43	8.2	55	5.2	P <0.01**
	Derbyshire	10	13.5	[illegible]	5.4	82	4.0	P <0.001

⁺Smoker = a child who smoked at least one cigarette a week.

†Experimental smoker = a child who had smoked at sometime but less than one cigarette a week.

*Test for significant association of cough and smoking habit. Chi-square 2 × 3 table.

**Smokers and experimental smokers combined to give chi-square on a 2 × 2 table.

SOURCE: Bewley, B.R. (9).

TABLE 12.—Prevalence (percent) of respiratory symptoms by sex and smoking habit in cohort of 3,898 20-year-olds followed since birth

History of Cigarette Smoking	Population		Winter Morning Cough Q.1(a)[1]		Cough Day or Night in Winter Q.1(b)[1]		Cough 3 Months in Winter Q.1(c)[1]		Winter Morning Phlegm Q.2(a)[1]		Phlegm Day or Night in Winter Q.2(b)[1]		Phlegm 3 Months in Winter Q.2(c)[1]		Persistent Cough and Plegm Q.1(c) +2(c)[1]	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Never smoked cigarettes	802	1093	1.6	4.0	5.2	6.5	1.5	3.2	4.8	5.2	6.4	3.9	3.7	3.2	0.9	1.9
Ex-smokers of cigarettes	101	57	3.0	1.8	7.1	10.5	3.0	1.8	11.0	1.9	10.2	9.1	6.0	0.0	2.0	0.0
Present smoker of cigarettes	1009	678	13.0	13.2	13.9	16.0	8.1	7.5	14.1	11.9	11.6	11.2	8.3	5.5	4.9	3.5
No data on cigarette smoking	92	48	8.7	11.8	9.1	18.8	4.5	0.0	0.0	6.7	4.8	0.0	4.8	0.0	4.8	0.0
All	2022	1876	7.7	7.4	9.8	10.2	5.0	4.7	9.9	7.6	9.3	6.7	6.2	3.9	3.0	2.4

[1] 1. (a) Do you usually cough first thing in the morning in the winter?
(b) Do you usually cough during the day or at night in the winter?
If "Yes" to either question 1(a) or (b)
(c) Do you cough like this on most days for as much as three months each winter?

2. (a) Do you usually bring up any phlegm (spit from the chest) first thing in the morning in the winter?
   (b) Do you usually bring up any phlegm (spit from the chest) during the day or at night in the winter?
   If "Yes" to either question 2(a) or (b)
   (c) Do you bring up phlegm (spit from the chest) on most days for as much as three months each winter?

SOURCE: Colley, J.R.T. (15).

TABLE 13.—Percentages of nonsmokers and smokers with abnormal test results in three North American cities, using combined reference values*

	Men						Women					
	Nonsmokers			Smokers			Nonsmokers			Smokers		
	AS (95)**	S (27)	Total (122)	AS (12)	S (115)	Total (236)	AS (145)	S (46)	Total (191)	AS (107)	S (98)	Total (205)
Upper limit +	1.6	0.2	1.8	1.8	1.8	2.6	2.1	0.6	2.4	1.7	1.7	2.4
Lower limit +	11.6	20.0	10.6	10.6	10.9	8.7	10.0	15.0	9.1	11.1	11.5	9.0
1. Abnormal test												
FEV-FVC	6	11	7	5	7	6	4	20	8	7	25	16
CV/VC	2	7	3	13	17	15	6	11	7	23	26	25
CC/TLC	2	7	3	20	32	26	8	17	10	20	29	25
ΔN/L	1	7	3	17	13	15	7	24	11	27	37	32
RV/TLC	6	11	7	9	9	9	8	9	8	11	13	12

*Reference values for nonsmokers derived from asymptomatic nonsmokers in the three cities.

**Numbers in parenthesis = number of subjects in each group.

+Upper and lower limits in the expected 5 percent abnormal results.

AS = asymptomatic; S = symptomatic

SOURCE: Buist, A.S. (11).

In a longitudinal study of elderly Edinburgh residents aged 61 to 90, Millne and Williamson found the prevalence of persistent cough and sputum production was significantly greater in smokers of both sexes than in their nonsmoking counterparts (40). Male prevalence rates were three times higher than those in females; however, no attempt was made to determine the relationship of respiratory symptoms to life-time tobacco exposure.

In summary, many recent studies demonstrate a higher frequency of respiratory symptoms in women who smoke as compared to women who do not smoke. This is true in surveys including children, adolescents, young adults, working age, and elderly women. The effect of cigarette smoking is related in terms of both the number of cigarettes and years smoked. The majority of studies indicate a greater prevalence of respiratory symptoms among men who smoke than among women who smoke; however, these differences often disappear when the study is carefully controlled for smoking history.

Smoking and Pulmonary Function

The insensitivity of cough and sputum production in the adult as a predictor of future development of COLD has been emphasized by Fletcher and Peto (29). Pulmonary function testing offers an objective method for measuring the adverse effects of smoking. However, current tests of pulmonary function display a marked variability between individuals and may not detect the development of COLD until irreversible damage of the lung has occurred. Also, none of the presently used pulmonary function tests can predict which of those individuals with slightly abnormal pulmonary function will progress to debilitating and life-threatening emphysema and chronic bronchitis. Becklake and Permutt have recently reviewed the objectives and problems of the tests of lung function commonly used for early detection of COLD (7).

A large number of studies have established a higher frequency of pulmonary functional abnormalities in smokers as compared to nonsmokers. These studies have examined (a) the relationship of smoking to abnormal tests of small airway function and (b) the relationship of smoking to measurements of standard spirometry. The majority of epidemiologic surveys investigating the prevalence of functional abnormalities in smokers have employed spirometric measurements, usually the forced expiratory volume (FEV) and vital capacity (VC). Measurements of airway resistance, diffusing capacity, lung volume, and nitrogen mixing have been used much less frequently.

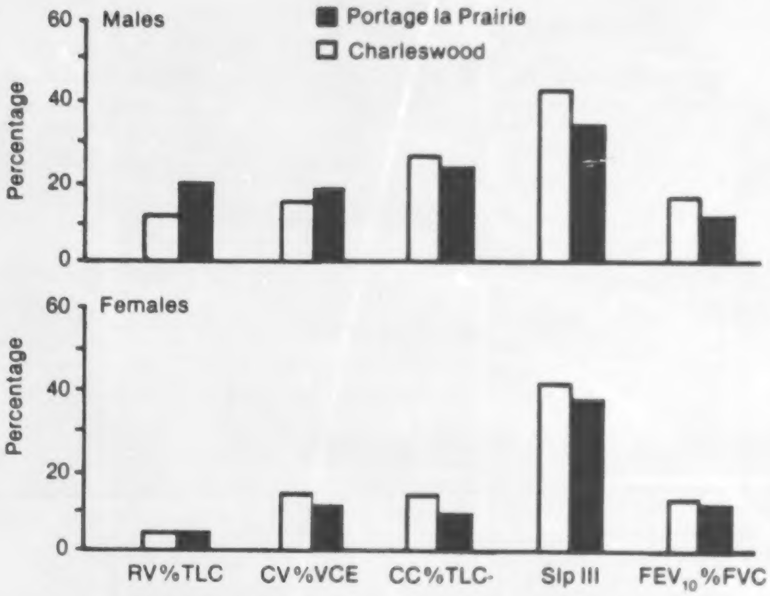

FIGURE 2.—Prevalence of lung function abnormalities among smokers in an urban (Charleswood) and a rural (Portage La Prairie) community

SOURCE: Manfreda, J. (39).

SMOKING AND "EARLY" FUNCTIONAL ABNORMALITIES

The most widely used measurements for detecting early change of chronic airflow obstruction are the single-breath nitrogen washout curve or a maximum forced expiratory volume curve.

A limited number of recent studies using tests of small airway function have included appreciable numbers of female subjects. They have demonstrated a higher frequency of abnormalities in tests of small airway function in smokers than in nonsmokers or ex-smokers. A definite dose-response relationship has been found in some of these studies but not in others (10,11,12). Table 13 shows the data from one of these studies (11). For all measures of small airway function, the frequency of abnormalities was higher among smokers than nonsmokers in both men and women. The frequency of abnormal measurements was considerably higher in female smokers than in male smokers except for closing capacity, in which equal proportions of male and female smokers performed abnormally. However, the frequency of abnormalities among female nonsmokers was also greater than among male nonsmokers. The authors speculate that the traditional view of chronic airflow obstruction as being predominantly a disease of males may be accurate only when male smokers outnumber female smokers and when males smoke more cigarettes than females. They suggest that when women's smoking habits become comparable to those of men, the effect on lung function may be similar.

Manfreda et al. used the single-breath nitrogen test in a large group of subjects in two Canadian cities (Figure 2) (39). Almost all smokers (85 percent) reported that they inhaled their cigarettes. Smokers had a greater prevalence of abnormalities than nonsmokers regardless of sex. The prevalence of abnormal values in women who smoke was slightly less than in male smokers.

In a volunteer population of 530 cigarette smokers attending an emphysema screening center, Buist and Ross found an equivalent frequency of abnormalities of the slope of phase III among male and female smokers of less than 20 cigarettes per day (Figure 3) with both sexes having significantly higher prevalence of abnormalities among smokers of more than 20 cigarettes per day (12). In the groups smoking more than 20 cigarettes a day, a greater proportion of females demonstrated abnormalities than males. However, the age composition of each group (male and female) was not identical.

A recent study of small airway function in 205 young volunteer smokers aged 18 to 25 has suggested that smoking may exert its effects at different anatomic locations in the lungs of men and women (21). All subjects smoked fairly heavily (more than 20 cigarettes per day) for a short period of time (average: 2.4 pack-years). Male smokers showed frequent abnormalities in tests of small airway function but female smokers did not exhibit these abnormalities. Both male and female smokers showed decreased forced expiratory flows at high lung volumes, suggesting the presence of large-airway dysfunction in young smokers. Male and female smokers differed significantly in their response to He-O_2 inhalation. Female smokers showed at least as great an improvement in forced expiratory flows with He-O_2 as did female nonsmokers. In contrast male smokers showed a much smaller response to the He-O_2 at high lung volumes. Thus, the predominant female response to habitual cigarette smoking appears to have been involvement of the large airways, but men who smoked appeared to have developed abnormalities in small airway function. The reason(s) for the differences in the data derived from this study and previously

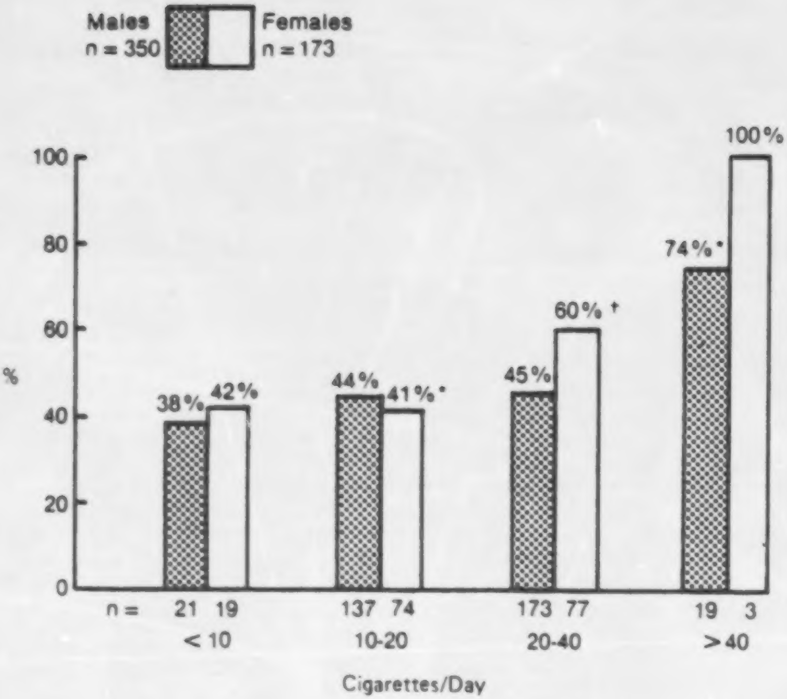

FIGURE 3.—Percentage of male and female cigarette smokers with an abnormal change in nitrogen concentration (ΔN_2) per liter according to their daily cigarette consumption

*Indicates a significant difference between groups using 20 to 40 cigarettes per day as the reference group ($P < 0.05$).

† Indicates significant differences between males and females ($P < 0.05$).

SOURCE: Buist, A.S. (12).

cited reports relating smoking to small airway dysfunction (11,12,39) is unclear.

In summary, a limited number of recent studies have demonstrated a higher frequency of abnormalities in tests of small airway function in female smokers as compared to female nonsmokers and ex-smokers. It is not clear whether these abnormalities are dose-related. Female smokers may have more frequent abnormalities in the slope of phase III than male smokers. Male smokers may have more frequent abnormalities in closing volume than female smokers. The meaning of these differences is unclear. One study has suggested that the earliest effects of smoking on lung function may occur in the large airways in women and small airways in men.

SMOKING AND VENTILATORY FUNCTION

The majority of studies examining the relationship of smoking to ventilatory capacity have used some measurement of forced expiratory volume. Most of these studies have focused on male populations and have found a close relationship between cigarette smoking and the presence of abnormal pulmonary function (2,6,16,20). Furthermore, the decrement in performance measured by simple spirometry is dose-related to the numbers of cigarettes smoked (6,16,20). Relatively few studies have included appreciable numbers of females.

Woolf examined pulmonary function in 500 women volunteers (65). Smokers demonstrated significantly lower values for FVC, FEV, FEF 25–75 percent, and specific conductance than nonsmokers and ex-smokers who had not smoked for over a year; this suggests that at least some abnormalities of pulmonary function are reversible with smoking cessation.

Higgins and Keller examined the relationship of smoking to seven derivatives of the forced vital capacity curve in 3,109 males and 3,256 females aged 10 and older (35). Nonsmokers performed better than smokers in both sexes. Values consistently decreased with increasing cigarette consumption. The largest differences were in FEV and FEF 25–75 percent.

Seltzer et al. examined the relationship of smoking to FVC in 65,086 white, black, and Asian subjects aged 20 to 79 who had attended a Kaiser-Permanente multiphasic health clinic (49). The authors found a significant reduction in FVC among white women who smoked as compared to nonsmoking white women. No such differences were found for black and Asian subjects, however. No explanation for this racial difference was apparent from their data.

In a study by Buist et al., the prevalence of abnormalities of FEV_1/FVC was higher in female smokers than nonsmokers (11). The frequency of abnormalities in FEV_1/FVC among female smokers was twice that of male smokers (Table 12). Gibson et al. examined the relationship of smoking to measurements of the forced vital capacity in 18,359 men and women in Australia (30). Nonsmokers had better lung functions than smokers. Among smokers of 10 or more cigarettes a day, men showed a greater decrement in lung function than women.

Burrows et al. examined the relationship of smoking to measurements of forced expiratory volume in 883 men and 1,166 women in Tucson, Arizona (13). Nonsmokers performed better

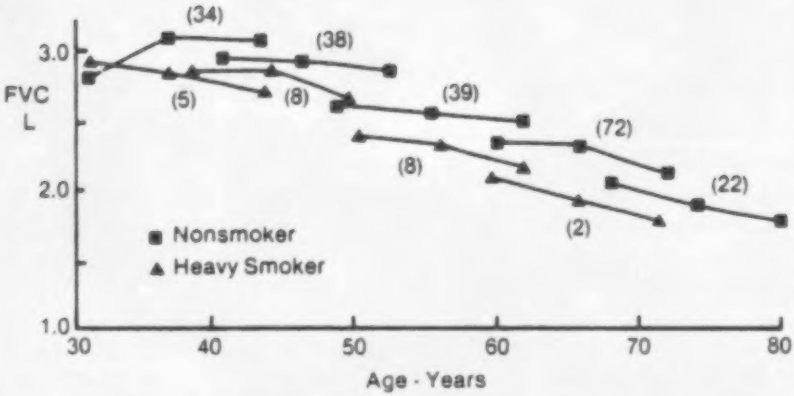

FIGURE 4.—Changes in forced vital capacity (FVC) by age in various female cohorts

Results have been standardized to 155 cm and are body temperature and pressure saturated (BTPS).

Numbers in parentheses are number in that cohort.

Heavy smokers are those who smoke 25 or more cigarettes per day.

SOURCE: Ferris, B.G., Jr. (23).

than ex-smokers or smokers, and ex-smokers performed better than smokers in both sexes. Smokers of more than 20 cigarettes per day performed worse than smokers of fewer than 20 cigarettes per day. There were no significant differences in the regression for FEV_1/FVC on pack years in men and women, suggesting that men and women with equivalent smoking habits have similar decrements in FEV_1/FVC.

The long-term effects of smoking on pulmonary function have been scrutinized in two prospective studies. In the Framingham study, 5,209 adults have been followed since 1948 with biennial examinations including measurements of forced vital capacity (3). Longitudinally, cigarette smokers showed a more rapid decline in forced vital capacity than nonsmokers. Men and women who continued to smoke had a more rapid decline in FVC than those who had stopped. The rate of decline in pulmonary function was appreciably steeper in male smokers than female smokers. The authors suggest that these differences could be due to differences in smoking habits.

In a longitudinal study of residents of Berlin, New Hampshire, Ferris examined the changes in pulmonary function by smoking status in the various age cohorts (23). Among females, heavy and moderate smokers had lower values for FVC and FEV_1 as compared to nonsmokers, and the values fell more

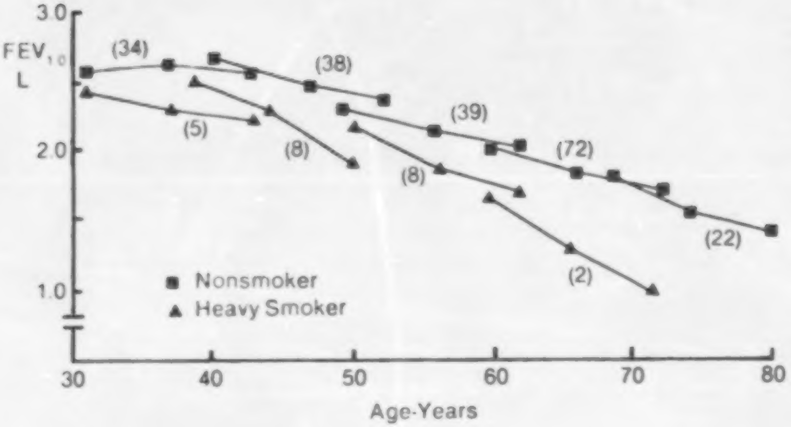

FIGURE 5.—Changes in forced expiratory volume in 1 second ($FEV_{1.0}$) by age in various female cohorts

Results have been standardized to 155 cm and are body temperature and pressure saturated (BTPS).

Numbers in parentheses are number in that cohort.

Heavy smokers are those who smoke 25 or more cigarettes per day.

SOURCE: Ferris, B.G., Jr. (23).

rapidly with age. These relationships for heavy smokers (25 or more cigarettes a day) are presented in Figures 4 and 5.

In summary, women smokers perform worse on spirometric testing than do female ex-smokers or nonsmokers. This relationship appears to be dose-related to the number of cigarettes smoked. The differential effects of smoking on pulmonary function in males and females is unclear. One study demonstrated that men and women with equivalent smoking habits have similar decrements in FEV_1/FVC. The long-term effect of smoking on pulmonary function has been evaluated in two studies which included appreciable numbers of females. Longitudinally, women who smoke show a more rapid decline in forced vital capacity than women who do not smoke. Women who continue to smoke have a more rapid decline in forced vital capacity than those who stop; however, men who continue to smoke have an even more rapid decline in pulmonary function than women who continue to smoke. The long-term relationship between respiratory symptoms and airflow obstruction in women is unknown. One large prospective study could not find a relationship between symptoms and the ultimate development of chronic airflow obstruction in men (29).

Summary

1. Recent statistics indicate a rising death rate due to chronic obstructive lung disease (COLD) among women. The data available demonstrate an excess risk of death from COLD among smoking women over that of nonsmoking women. This excess risk is much greater for heavy smokers than for light smokers.

2. Women's total risk of COLD appears to be somewhat lower than men's, a difference which may be due to differences in prior smoking habits.

3. The prevalence of chronic bronchitis varies directly with cigarette smoking, increasing with the number of cigarettes smoked per day.

4. There is conflicting evidence regarding differences in the prevalence of chronic bronchitis in women and men. Several recent studies suggest that there is no significant difference in the prevalence of chronic bronchitis between male and female smokers. This may be the result, however, of increasingly similar smoking behavior of women and men.

5. The presence of emphysema at autopsy exhibits a dose-response relationship with cigarette smoking during life.

6. There is a close relationship between cigarette smoking and chronic cough or chronic sputum production in women, which increases with total pack-years smoked.

7. Women current smokers have poorer pulmonary function by spirometric testing than do female ex-smokers or nonsmokers, a relationship which is dose-related to the number of cigarettes smoked.

References

(1) AMERICAN COLLEGE OF CHEST PHYSICIANS. AMERICAN THORACIC SOCIETY. Pulmonary terms and symbols. A report of the ACCP-ATS Joint Committee on Pulmonary Nomenclature. Chest 67: 583–593, 1975.

(2) ASHFORD, J.R., BROWN, S., DUFFIELD, D.P., SMITH, C.S., FAY, J.W.J. The relation between smoking habits and physique, respiratory symptoms, ventilatory function, and radiopneumoconiosis amongst coal workers at three Scottish collieries. British Journal of Preventative and Social Medicine 15: 106–117, 1961.

(3) ASHLEY, F., KANNEL, W.B., SORLIE, P.D., MASSON, R. Pulmonary function: Relation to aging, cigarette habit and mortality. The Framingham Study. Annals of Internal Medicine 82(5): 739–745, 1975.

(4) AUERBACH, O., GARFINKEL, L., HAMMOND, E.C. Relation of smoking and age to findings in lung parenchyma: A microscopic study. Chest 65(1): 29–35, 1974.

(5) AUERBACH, O., HAMMOND, E.C., GARFINKEL, L., BENANTE, C. Relation of smoking and age to emphysema. Whole-lung section study. New England Journal of Medicine 286(16): 853–857, 1972.

(6) BALCHUM, O.J., FLETON, J.S., JAMISON, J.N., GAINES, R.S., CLARKE, D.R., OWAN, D. The Industrial Health Committee, The

Tuberculosis and Health Association of Los Angeles County. A survey of chronic respiratory disease in an industrial city. Preliminary results. American Review of Respiratory Disease 86(5): 675–685, November 1962.

(7) BECKLAKE, M., PERMUTT, S. The Lung in Transition Between Health and Disease. New York, Marcel Dekker, Inc., 1979, pp. 345–387.

(8) BEST, E.W.R., JOSIE, G.H., WALKER, C.B. A Canadian study of mortality in relation to smoking habits: A preliminary report. Canadian Journal of Public Health 52: 99–106, March 1961.

(9) BEWLEY, B.R., BLAND, J.M. Smoking and respiratory symptoms in two groups of school children. Preventative Medicine 5: 63–69, 1976.

(10) BUIST, A.S., FLEET, L.V., ROSS, B.B. A comparison of conventional spirometric tests and the test of closing volume in an emphysema screening center. American Review of Respiratory Disease 107: 735–743, 1973.

(11) BUIST, A.S., GHEZZO, H., ANTHONISEN, N.R., CHERNIAK, R.M., DUCIC, S., MACKLEM, P.T., MANFREDA, J., MARTIN, R.R., MCCARTHY, D., ROSS, B.B. Relationship between the single breath N_2 test and age, sex, smoking habit in the North American cities. American Review of Respiratory Disease 120: 305–318, 1979.

(12) BUIST, A.S., ROSS, B.B. Quantitative analysis of the alveolar plateau in the diagnosis of early airway obstruction. American Review of Respiratory Disease 108: 1078–1087, 1973.

(13) BURROWS, B., KNUDSON, R.J., CLINE, M.B., LEBOWITZ, M.D. Quantitative relationships between cigarette smoking and ventilatory function. American Review of Respiratory Disease 115: 195–205, 1977.

(14) CEDERLOF, R., FRIBERG, L., HRUBEC, Z., LORIER, V. The Relationship of Smoking and Some Social Covariables to Mortality and Cancer Morbidity. A Ten Year Follow-up in a Probability Sample of 55,000 Swedish Subjects Age 18–69. Part 1 and 2. Stockholm, Sweden. The Karolenska Institute, Department of Environmental Hygiene, 1975, 201 pp.

(15) COLLEY, J.R.T., DOUGLAS, J.W.B., REID, D.D. Respiratory disease in young adults: Influence of early childhood lower respiratory tract illness, social class, air pollution, and smoking. British Medical Journal 3: 195–198, July 1973.

(16) DENSEN, P.M., JONES, E.W., BASS, H.E., BREUER, J., REED, E. A survey of respiratory disease among New York City postal and transit workers. 2. Ventilatory function tests results. Environmental Research 2(4): 277–296, July 1969.

(17) DOLL, R., GRAY, R., PETO, R. Mortality in relation to smoking: Observations on female doctors. (Unpublished manuscript)

(18) DOLL, R., PETO, R. Mortality in relation to smoking: 20 years observations on male British doctors. British Medical Journal 2(6051): 1525–1536, December 25, 1976.

(19) DUNN, J.E., LINDEN, G., BRESLOW, L. Lung cancer mortality experience of men in certain occupations in California. American Journal of Public Health 50(10): 1475–1487, October 1960.

(20) EDELMAN, N.H., MITTMAN, C., NORRIS, A.H., COHEN, B.H., SHOCK, N.W. The effects of cigarette smoking upon spirometric performance of community dwelling men. American Review of Respiratory Disease 94(3): 421–429, September 1966.

(21) ENJETTI, S., HAZELWOOD, B., PERMUTT, S., MENKES, H., TERRY, P. Pulmonary function in young smokers. Male-female differences. American Review of Respiratory Disease 118: 667–675, 1978.

(22) FERRIS, B.G., JR. Chronic bronchitis and emphysema. Medical Clinics of North America 57: 637-649, 1973.

(23) FERRIS, B.G., JR. Smoking and lung function: Epidemiological evidence. Proceedings of the Third World Conference on Smoking and Health 2. U.S. Department of Health, Education, and Welfare. Public Health Service. National Institutes of Health, p. 115-129, 1975.

(24) FERRIS, B.G., JR., CHEN, H., PULEO, S., MURPHY, R.L.H., JR. Chronic nonspecific diseases in Berlin, New Hampshire, 1967-1973. American Review of Respiratory Disease 113: 475-485, 1976.

(25) FERRIS, B.G., JR., HIGGINS, I.T.T., HIGGINS, J.M., PETERS, J.M., VAN GANSE, W.F., GOLDMAN, M.W. Chronic nonspecific respiratory disease, Berlin, New Hampshire, 1961-1967: A cross-sectional study. American Review of Respiratory Disease 104: 232-244, 1971.

(26) FERRIS, B.G., JR., HIGGINS, I.T.T., PETERS, J.M., VAN GANSE, W.F., GOLDMAN, M. Chronic nonspecific respiratory disease, Berlin, New Hampshire, 1961-1967: A cross-sectional study. American Review of Respiratory Diease 104: 232-244, 1971.

(27) FLETCHER, C.M. (Editor). Terminology, definitions, classification of chronic pulmonary emphysema and related conditions. A report of the conclusions of a Ciba Guest Symposium. Thorax 14: 286-299, 1959.

(28) FLETCHER, C.M., JONES, N.L., BURROWS, B., NIDEN, A.H. American emphysema and British bronchitis. A standardized comparative study. American Review of Respiratory Disease 90: 1-13, 1964.

(29) FLETCHER, C., PETO, R. The natural history of chronic airflow obstruction. British Medical Journal 1: 1645-1648, 1977.

(30) GIBSON, J., GALLAGHER, H., JOHANSON, A., WEBSTER, I. Lung function in an Australian population. 2. Spirometric performance and cigarette smoking habits. Medical Journal of Australia 1: 354-358, 1979.

(31) HAMMOND, E.C. Smoking in relation to the death rates of one million men and women. In: Haenszel, W. (Editor). Epidemilogical Approaches to the Study of Cancer and other Chronic Diseases. National Cancer Institute Monograph 19. U.S. Department of Health, Education, and Welfare, U.S. Public Health Service, National Cancer Institute, January 1966, pp. 127-204.

(32) HAMMOND, E.C., HORN, D. Smoking and death rates—Report on forty-four months of follow-up on 187,783 men. I. Total mortality. Journal of the American Medical Association 166(10): 1159-1172, March 8, 1958.

(33) HIGGINS, I.T.T. Respiratory symptoms, bronchitis and disability in a random sample of an agricultural population. British Medical Journal 2: 1198-1203, 1957.

(34) HIGGINS, I.T.T., COCHRAN, J.B. Respiratory symptoms, bronchitis and disability in a random sample of an agricultural community in Dumfrieshire. 39: 296-301, 1958.

(35) HIGGINS, M.W., KELLER, J.B. Seven measures of ventilatory lung function. American Review of Respiratory Disease 108: 258-272, 1973.

(36) HUBTI, E. Prevalence of respiratory symptoms, chronic bronchitis and pulmonary emphysema in a Finnish rural population. Field survey of age 40-64 in the Harjavolta area. Aeta (Supplement) 61: 11, 1965.

(37) KAHN, H.A. The Dorn study of smoking and mortality among U.S. veterans. Report on 8 and one-half years of observation. In: Haenszel, W. (Editor). Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases. National Cancer Institute Monograph 19. U.S. Department of Health, Education, and Welfare, Public Health Service,

National Cancer Institute, January 1966, pp. 1-125.

(38) LEIBOWITZ, M., BURROWS, B. Quantitative relationships between cigarette smoking and chronic productive cough. International Journal of Epidemiology 6: 107-113, 1977.

(39) MANFREDA, J., NELSON, N., CHERNIACK, R.M. Prevalence of respiratory abnormalities in a rural and an urban community. American Review of Respiratory Disease: 117: 215-226, 1978.

(40) MILLNE, J., WILLIAMSON, J. The relationship of respiratory function tests to respiratory symptoms and smoking in older people. Respiration 29: 206-213, 1972.

(41) MUELLER, RE., KEBLE, D., PLUMMER, J., WALKER, S.H. The prevalence of chronic bronchitis, chronic airway obstruction, and respiratory symptoms in a Colorado city. American Review of Respiratory Disease 103: 209-228, 1971.

(42) NATIONAL CENTER FOR HEALTH STATISTICS. Vital Statistics of the United States, 1960-1977. U.S. Department of Health, Education, and Welfare, Public Health Service, Office of Health Policy, Research and Statistics, National Center for Health Statistics.

(43) OSWALD, N.C., MEDVEL, V.C. Chronic bronchitis: the effect of cigarette smoking. Lancet 2: 843-844, October 22, 1955.

(44) PAYNE, M., KJELSBERG, M. Respiratory symptoms, lung function, and smoking habits in an adult population. American Journal of Public Health 54: 261-277, 1964.

(45) RAWBONE, R., KEELING, C., JENKINS, A., GUZ, A. Cigarette smoking among secondary school children in 1975. Journal of Epidemiology and Community Health 32: 53-58, 1978.

(46) REMINGTON, J. Chronic bronchitis, smoking and social class. A study among working people in the towns of East and Mid Cheshire. British Journal of Disease of the Chest 63(4): 193-205, 1969.

(47) RUSH, D. Changes in respiratory symptoms related to smoking in a teenage population: The results of tow linked surveys separated by one year. International Journal of Epidemiology 5(2): 173-178, 1976.

(48) RYDER, R., DUNNILL, M., ANDERSON, J. A quantitative study of bronchial mucous gland volume, emphysema and smoking in a necropsy population.

(49) SELTZER, C.C., SIEGELAUB, A.B., FRIEDMAN, G.D., COLLEN, M.F. Differences in pulmonary function related to smoking habits and race. American Review of Respiratory Disease 110(5): 598-608, November 1974.

(50) SPAIN, D.M., SIEGEL, H., BRADES, V.S. Emphysema in apparently healthy adults. Journal of the American Medical Association 224: 322-325, 1973.

(51) TAGER, I.B., SPEIZER, F.E. Risk estimates for chronic bronchitis in smokers: A study of male-female differences. American Review of Respiratory Diseases: 113: 619-625, 1976.

(52) THURLBECK, W.M. Aspects of chronic airflow obstruction. Chest 72: 341-349, 1977.

(53) THURLBECK, W.M. Chronic airflow obstruction in lung disease. V. Major Problems in Pathology. Philadelphia, W.B. Sanders Co., 1976, pp. 235-287.

(54) THURLBECK, W.M., RYDER, R., STERNLY, N. A comparative study of severity of emphysema in necropsy population in three different countries. American Review of Respiratory Disease 109: 239-248, 1974.

(55) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking. A Public Health Service Review: 1967. U.S. Department of Health

Service, Health Services and Mental Health Administration. DHEW Publication No. 1969, Revised, January 1968, 227 pp.

(56) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking, 1968. Supplement to the 1967 Public Health Service Review. U.S. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration. DHEW Publication No. 1969, 1968, 117 pp.

(57) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking, 1969. Supplement to the 1967 Public Health Service Review. U.S. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration. DHEW Publication No. 1969-2, 1969, 98 pp.

(58) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking. A Report of the Surgeon General. 1971. U.S. Department of Health Services and Mental Health Administration. DHEW Publication No. 71-7513, 1971, 458 pp.

(59) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking. A Report of the Surgeon General: 1972. U.S. Department of Health Services and Mental Health Administration. DHEW Publication No. (HSM) 72-7516, 1972, 158 pp.

(60) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking. 1973. U.S. Department of Health, Education, and Welfare, Public Health Services, Health Services and Mental Health Administration. DHEW Publication No. (HSM) 73-8704, 1973, 249 pp.

(61) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking: 1974. U.S. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration. DHEW Publication No. (CDC) 74-8704, 1974, 124 pp.

(62) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking: 1975. U.S. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration. DHEW Publication No. (CDC) 76-8704, 1975, 235 pp.

(63) U.S. PUBLIC HEALTH SERVICE. Smoking and Health. Report of the Advisory Committee to the Surgeon General of the Public Health Service. U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control. PHS Publication No. 1103, 1964, 387 pp.

(64) U.S. PUBLIC HEALTH SERVICE. Smoking and Health. A report of the Surgeon General, U.S. Department of Health, Education, and Welfare, Public Health Service. Office of the Assistant Secretary for Health, Office on Smoking and Health. DHEW Publication No. (PHS) 79-50066, 1979, pp. 1251.

(65) WOOLF, C.R. Clinical findings, sputum examinations, and pulmonary function tests related to the smoking habits of 500 women. Chest 66: 652-659, 1974.

(66) WOOLF, C., SUERO, J. The respiratory effects of regular cigarette smoking in women. American Review of Respiratory Disease. 103: 26-37, 1971.

INTERACTION BETWEEN SMOKING AND OCCUPATIONAL EXPOSURES

The 1979 Surgeon General's Report on the health consequences of smoking (18) examines the interaction of smoking and occupational exposure. Ways in which smoking may interact with the occupational environment are described and examples of these interactions are discussed. Briefly, these types of interaction are:

1. Tobacco products may serve as vectors by becoming contaminated with toxic agents found in the workplace, thus facilitating entry of the agent by inhalation, ingestion, and/or skin absorption of the agent.
2. Workplace chemicals may be transformed into more harmful agents by smoking.
3. Certain toxic agents in tobacco products and/or smoke may also inhabit the workplace, thus increasing exposure to the agent.
4. Smoking may contribute to an effect comparable to that which can result from exposure to toxic agents found in the workplace, thus causing an additive biological effect.
5. Smoking may act synergistically with toxic agents found in the workplace to cause a much more profound effect than that anticipated simply from the separate influences of the agent and smoking added together.
6. Smoking may contribute to accidents in the workplace.

Although few of the studies discussed in the 1979 Surgeon General's Report included enough women to adequately determine the health risks of smoking and the occupational environment, it is reasonable to hypothesize that women with the same occupational exposure and smoking behavior as men would develop health effects similar to those demonstrated in men. However, the interaction of smoking and the occupational environment and its effect on women differs in at least two ways:

First, smoking patterns among women are different from those among men—women are less likely to smoke, and if they do, they smoke fewer cigarettes per day, inhale less, and are more likely to smoke lower "tar" and nicotine cigarettes (7,14,18). Second, smoking and occupational exposure may adversely affect the fetus or the health of the mother during pregnancy. Smoking and occupational exposure may also interact with methods of contraception chosen by women.

This chapter reviews each of these reasons for a differential health impact on men and women and examines two occupational exposures where interactions with smoking have been clearly demonstrated for women workers.

TABLE 1.—Smoking habits of working women by title and industry

Industry	Percent of Current Female Labor Force*	Percent			
		Non-Smokers	Ex-Smokers	Present Smokers	
				≤ 1 pack per day	≥ 1 pack per day
Professionals					
Health	4.4	51.2	16.6	25.2	6.9
Teachers	6.8	63.5	14.0	19.8	2.7
Other	4.6	53.4	15.1	24.0	7.5
Managerial, incl. office, rest., sales, administrator	6.7	42.7	16.4	28.0	12.1
Sales	6.2	46.0	16.2	30.0	8.0
Clerical					
Bookkeepers	4.6	53.1	12.2	26.5	8.2
Office machine operators	1.3	52.8	15.7	23.1	8.4
Secretaries	13.3	52.0	14.7	26.3	7.0
All other	14.2	50.6	13.6	27.5	8.3
Crafts	2.4	46.4	13.1	31.8	8.6
Operatives	11.8	52.8	10.1	31.6	5.5
Service					
Cleaning	2.5	51.9	12.8	31.2	4.1
Food	6.6	40.0	13.4	39.8	6.8
Health	6.9	52.1	10.5	32.2	5.2
Private Household Workers	2.8	62.4	10.1	24.7	2.8

*Figures are subject to sampling errors and may therefore not agree with those in other tables.

SOURCE: National Center for Health Statistics (6).

Smoking Patterns in Women

The male–female differences in smoking behavior and the change in patterns of smoking behavior in women over time are reviewed in other sections of this report. It is important, however, to consider the impact of these trends when evaluating the interaction of smoking and the environment. Regular cigarette smoking is a behavior that usually begins between the ages 12 and 25 (18). It is unusual to begin regular smoking after the age

TABLE 2.—**Estimates of the percentage of current, regular cigarette smokers, adults ages 20 years and over, according to labor force status and occupation and sex, U.S., 1976**

	Female			Male		
	Total 20+	20–44	45–64	Total 20+	20–44	45–64
Total	32.0	36.9	34.8	41.9	47.6	41.3
Currently employed	35.9	37.0	36.1	43.4	46.8	39.7
White collar total	34.3	33.8	36.9	36.6	38.6	35.3
Professional technical and kindred	29.1	28.6	32.7	30.0	31.1	29.9
Managers & administrators except farm	41.6	42.7	40.8	41.0	46.4	36.1
Sales workers	38.1	37.0	42.6	39.9	42.6	38.0
Clerical & kindred workers	34.8	34.7	36.0	40.4	40.1	44.2
Blue collar total	39.0	43.7	33.6	50.4	54.1	44.3
Craftsmen & kindred workers	40.5	46.9	35.6	48.0	52.1	41.6
Operatives and kindred workers	37.6	42.5	31.2	52.3	55.3	46.2
Laborer, except farm	56.3	52.6	*	53.7	56.9	51.7
Service	39.0	42.8	37.2	47.2	51.1	44.8
Farm	32.2	51.0	*	36.9	45.4	35.0
Unemployed	40.0	41.0	39.2	56.8	59.9	53.8
Usual activity—homemaking	29.0	37.1	32.2	NA	NA	NA

NOTE: Unknown if ever smoked excluded from calculation.
*Figure does not meet standards of reliability or precision.
SOURCE: National Center for Health Statistics (6).

of 25 (7). In a cohort of individuals born in the same year, a certain percentage of them will begin smoking by age 25. The prevalence of smoking in any birth cohort after age 25 is predominantly determined by the rate at which people stop smoking or die. The prevalence changes over time for each 10 year birth cohort since 1910 for both men and women are presented in the part of this report titled Patterns of Cigarette Smoking.

Women first began smoking cigarettes in large numbers immediately before and during the Second World War (18). Thus, the observed upswing in smoking among women occurred 25 to 30 years after that among men. The birth cohorts with the high-

TABLE 3.—Occupational distribution of men and women, 1978, by percent of each sex employed in each category

	Women	Men
Professional, Technical	15.6	14.7
Sales	6.9	5.9
Clerical	34.6	6.2
Operatives & Transport	11.8	17.7
Service	20.7	8.7
All Other	2.5	11.7
Crafts	1.8	21.1
Managers	6.1	14.0
Total	100	100

SOURCE: Rones, P. (14).

est peak smoking prevalence were born from 1910 to 1930 (men) and from 1920 to 1950 (women). As these cohorts with high prevalence of smoking grow older, they replace cohorts with lower smoking prevalence. Since both occupational diseases and smoking related illnesses increase separately with age, any interaction between the two also could be expected to increase with age. Men in the birth cohort from 1910 to 1930 are now in the age range at which a high incidence of disease would be expected, while those women born from 1920 to 1950 are just beginning to enter the ages at which there is a high prevalence of disease. As a result, the adverse effects of smoking and occupational exposure would be expected to occur more frequently in men, reflecting this difference in the age of the average male and female smoker. This "cohort effect" might lead to the erroneous conclusion that women are protected from occupation–smoking interactions, just as it has been used to suggest that women are protected from the lung cancers induced by cigarette smoking.

A second difference between male and female smoking habits which must be considered is the prevalence of smoking by occupation. Table 1 shows that the prevalence of smoking is reasonably uniform among women employed in many different occupations (the exceptions are education and household area workers with low prevalence and food area workers with high prevalence). There is not the marked difference in smoking habits between female blue collar and white collar workers that has been observed in men (13) (Table 2). A slightly lower prevalence of smoking among professional women compared to other white collar workers occurs similar to that seen in men (7).

The section on behavior in this report discusses the smoking habits of several groups of health professionals. It shows that women physicians and psychologists smoke more heavily than their male counterparts. Thus, the relative levels of smoking observed in the two sexes are reversed for these two occupational groups in comparison to the general population (14). Nurses also have been shown to have a much higher prevalence of smoking than women of the same age in the general population (18). A final notable difference is that, among women, smoking prevalence does not show the same marked inverse correlation with socioeconomic status (7). The reasons for these differences are beyond the scope of this section. However, an understanding of them forms part of the background for any discussion of the interaction of smoking and occupational exposures among women.

Patterns of Employment

The percentage of women in the United States work force is steadily growing. In 1973 women represented 38.4 percent of the United States work force and in 1978 that percentage had risen to 41.2 percent (15).

Approximately 39 million women are employed outside the home. Table 3 clearly indicates that the distribution of women in the labor force by category of work does not parallel that of men. Women are more likely than men to be employed in the clerical and service categories. Men are more likely to be employed in the management, crafts and operatives/transport categories than women. Table 4 lists the number of women employed in a wide variety of occupations, including many of those traditionally believed to be hazardous for men. In spite of this diversity, the bulk of women are employed in a narrow range of jobs. Over one-third of women in the paid labor force are employed in one of the 10 job categories listed in Table 5. All of these categories have been traditional employment areas for women. Thus, the recent gains by women in employment opportunity have not yet had a substantial impact on the actual distribution patterns of the female labor force. If a shift does occur in employment patterns involving greater proportions of women in occupations with significant exposures, we would expect a cohort effect to be apparent in the development of occupational illness. That is, those women entering hazardous occupations traditionally limited to male workers would be expected to be women newly entering the work force and, thus, predominantly in the younger age groups. As these cohorts age, the duration of both occupational and smoking exposures would increase. It is only after these newer cohorts reach the ages where disease is prevalent that we would be able to observe the full

TABLE 4.—Number of women in the current workforce, classified by occupation (1978)

Occupation	# of Women in Thousands
White-collar workers	24,594
Professional & Technical	6,083
Biological scientists	22
Chemists	17
Nurses, dieticians, & therapists	1,255
Health technologists and technicians	353
Engineering and science technicians	132
Painters and sculptors	83
Photographers	13
Managers and administrators, except farm	2,365
Sales workers	2,666
Sales clerks, retail trade	1,672
Clerical workers	13,456
Bookkeepers	1,660
Cashiers	1,222
Secretaries	3,561
Typists	1,009
Blue-collar workers	5,770
Craft and kindred workers	694
Printing craft workers	91
Upholsterers	14
Operatives, except transport	4,317
Assemblers	606
Bottling and canning operatives	25
Checkers, examiners, and inspectors; manufacturing	359
Clothing ironers and pressers	101
Cutting operative, n.e.c.	84
Dressmakers, except factory	113
Drillers, earth	2
Dry wall installers and lathers	1
Filers, polishers, sanders and buffers	38
Furnace tenders, smelters, and pourers, metal	3
Garage workers, and gas station attendants	20
Blue-collar workers—cont'd	
Laundry and dry cleaning operatives, n.e.c.	118
Meat cutters and butchers, except manufacturing	13
Meat cutters and butchers, manufacturing	33
Mine operatives, n.e.c.	4
Mixing operatives	3
Packing and wrappers, excluding meat and produce	422
Painters, manufactured articles	30
Photographic process workers	48
Precision machine operatives	43
Drill press operatives	15
Grinding machine operatives	10
Lathe and milling machine operatives	11
Punch and stamping press operatives	47
Sawyer	14
Sewers and stitchers	772
Shoemaking machine operatives	60
Furnace tenders and stokers, except metal	1
Textile operatives	224
Spinners, twisters, and winders	100
Welders and flame cutters	41
Winding operatives, n.e.c.	37
All other operatives, except transport	1,062
Transport equipment operatives	258
Nonfarm laborers	492
Service workers	8,037
Private households	1,135
Child care workers	477
Cleaners and servants	514
Housekeepers	117
Service workers, except households	6,901
Cleaning workers	858

Table 4 (continued)

Occupation	# of Women in Thousands
Service workers—cont'd.	
Lodging quarters cleaners	174
Building interior cleaners, n.e.c.	462
Janitors and sextons	222
Food service workers	2,951
Bartenders	111
Waiters' assistants	45
Cooks	678
Dishwashers	82
Food counter and fountain workers	397
Waiters	1,252
Food service workers, n.e.c.	384
Health service workers	1,660
Dental assistants	128
Health aides, excluding nursing	238
Nursing aides, orderlies, and attendants	902
Health service workers—cont'd.	
Practical nurses	390
Personal service workers	1,302
Attendants	175
Barbers	11
Child care workers	103
Hairdresser and cosmetologists	483
Housekeepers, excluding private households	—
Welfare service aides	92
Protective service workers	115
Firefighters	1
Guards	53
Police and detectives	28
Sheriffs and bailiffs	3
Farm workers	509
TOTAL—	38,910

NOTE: n.e.c. is an abbreviation for "not elsewhere classified" and designates broad categories of occupations that cannot be more specifically identified.
SOURCE: U.S. Department of Labor (17).

impact of occupational exposures (or their interactions with smoking) on the health of women.

Because of this cohort effect, any failure to demonstrate an excess risk of a given occupational exposure in women must be interpreted with considerable caution. It may mean only that the women exposed were too young and the exposure too brief for illness to have yet developed. This caution is doubly important for those attempting to demonstrate an interaction between occupational exposure and smoking on the development of disease in women. Thus, little comfort can be taken from the current low prevalence of occupational disease in women. It is reasonable to expect that any movement of large numbers of women into hazardous occupations will be followed, after an appropriate time lag, by a dramatic increase in the prevalence of occupational illness in women.

The Reproductive Role

A third reason for examining the effects of occupational exposures in women separately from those in men is the difference

TABLE 5.—**Most common female job categories, by percentage of the female work force employed**

Job	Percent of Female Work Force	Job	Percent of Female Work Force
Secretary	8.5	Private Household Worker	2.9
Retail Sales Clerk	4.3	Registered Nurse	2.8
Bookkeeper	4.3	Elementary School Teacher	2.8
Waitress	3.2	Typist	2.6
		Cleaning Workers	2.2
Cashier	3.1	Sewer & Stitcher	2.0

SOURCE: Rones, P. (14).

in their reproductive roles. Toxic occupational exposures in both men and women can reduce fertility and increase frequency of teratogenic effects (see Table 6). In addition, however, the 9-month duration of gestation provides many opportunities for the fetus to share any adverse toxic exposure of its mother. These risks may interact with the well-established risks of cigarette smoking during pregnancy discussed elsewhere in this report. Table 6 provides a list of hazardous substances in the work environment, some of which are suspected of having effects on reproduction.

Another specific concern for women is that of contraception. Substantial numbers of women in the United States use oral contraceptives (18). These drugs have been shown to interact with cigarette smoking to produce a greatly increased risk of cardiovascular disease, as discussed in this report. In addition, it is possible that oral contraceptives may interact in an adverse manner with physical or chemical agents found in the work place, or that the combination of smoking, occupational exposure, and oral contraceptive use may bear special risks. The answers to those questions can be found only through the study of populations of working women.

One study approached this issue by examining the health status of women involved in the manufacture of oral contraceptives. Poller, et al. have shown that women working in the manufacture of oral contraceptives absorb enough of the drugs to influence the clotting mechanism as well as alter menstrual function (12). Unfortunately, the risk of cardiovascular disease—and the effects of smoking in relation to it—could not be estimated in this population. Because of the established excess risk of cardiovascular disease from concurrent smoking and oral contraceptive use, examination of cardiovascular risk in this group would be of interest.

The preceding discussion presents several areas where female-male differences may significantly limit the direct applicability of the results of male smoking studies to the female population. These areas of potential difference present research questions that justify significant, ongoing research activities.

Specific Interactions Between Occupational Exposure and Smoking

A review of all the potential risks of occupational exposure for women is beyond the scope of this section. Table 6 lists a number of agents found in the occupational environment and their observed organ toxicity. Table 7 presents selected pulmonary irritants and sensitizers in specific occupational settings in relation to the number of women employed in those settings.

There is little specific data on the health effects of a given occupational exposure in women. Two clear exceptions exist—exposure to asbestos and to cotton dust. The data from studies of women exposed to these two compounds provide examples of established interactions between smoking and occupational exposure in women.

ASBESTOS

Selikoff, et al. prospectively followed a group of 370 male asbestos insulation workers. They demonstrated a multiplicative effect of asbestos exposure and cigarette smoking on the risk of development of lung cancer (4,13). Workers who smoked cigarettes developed lung cancer at a rate 92 times that of non-exposed nonsmokers. They observed no deaths from lung cancer among 87 nonsmokers, and 24 deaths from bronchogenic cancer among 283 regular smokers, a number well in excess of the 3 deaths expected. Newhouse, et al. followed a cohort of 900 women first employed between 1936 and 1942 in an asbestos factory making both textiles and insulation materials (2,10,11). They analyzed the group's mortality experience between first employment and 1968, with a minimum of 26 years' follow-up. There was an excess overall mortality partly accounted for by deaths from cancer, observed even among those who worked in jobs with low-to-moderate exposure to asbestos. An excess of cancer of the lung and pleura was found among those who were severely exposed and who had worked less than 2 years. In the group with severe exposure for more than 2 years in the factory, excess deaths from cancer of the lung, pleura, and non-

TABLE 6.—Chart of toxins and effects

AGENTS	DEFINITIVE SYSTEMIC TOXICITY						SPECIAL EFFECTS	SUSPECTED REPRODUCTIVE EFFECTS					SUGGESTED BIOLOGICAL TESTING	AIR STANDARDS	
	Neurologic	Hepatic	Renal	Hematologic	Cardiovascular	Pulmonary		Carcinogenesis	Reduced Fertility	Spontaneous Abortion	Mutagenesis	Teratogenesis		OSHA (existing)	NIOSH (proposed)
Heavy metals															
Cadmium		•	•	•	•	•		•	•			•	Urine cadmium Quant. low molecular weight proteins	fume 0.1 mg/M^3 dust 0.2 mg/M^3	40μg/M^3
Lead	•		•	•					•	•		•	Blood lead Blood zinc protoporphyrin Urine ALA Urine coproporphyrin	inorganic 0.2 mg/M^3 (Proposed 0.1 mg/M^3)	inorganic 0.1 mg/M^3 and Blood Lead 60μg/100gms
Mercury	•		•							•		•	Blood mercury Urine mercury	inorganic 0.1 mg/M^3 organic 0.01 mg/M^3	inorganic 0.05 mg/M^3
Organic solvents															
Benzene (benzol)	•			•				•				•	Urine phenols CBC	10 ppm (emergency proposal 1 ppm)	1 ppm
Halogenated hydrocarbons															
2 chlorobutadiene (chloroprene)	•	•	•	•		•		•	•	•	•		Liver function test	25 ppm	1 ppm
Dibromochloropropane		•	•					•	•				Sperm count Serum testosterone	10 ppb (emergency proposed)	10 ppb
Epichlorohydrin		•	•			•			•				Liver function test	20 mg/M^3	2 mg/M^3
Ethylene dibromide		•	•			•		•	•					20 ppm	1 mg/M^3
Polychlorinated biphenyls (PCB's)		•		•			Chloracne	•	•	•			Blood analysis Adipose tissue Serum transaminase	Compounds with 42% chlorine 1 mg/M^3 54% chlorine 0.5 mg/M^3	1 μg/M^3
Tetrachloroethylene (Perchloroethylene)	•	•				•		•			•	•		100 ppm	50 ppm
Vinyl chloride	•	•			•		Acroosteolysis	•			•		Liver function test	1 ppm	1 ppm
Hypoxic agents															
Carbon monoxide	•			•	•							•	Carboxy hemoglobin	50 ppm	35 ppm
Anesthetic gases															
Halogenated gases e.g. halothane methoxy fluorane	•	•						•	•	•	•	•		not specified	Halogenated anesthetics 2 ppm based on weight of specific gas sampled not >1 hour
Pesticides															
Carbaryl	•								•					50 mg/M^3	50 mg/M^3
Chlorinated hydrocarbons (e.g. chlordane)				•				•			•		Adipose tissue analysis Blood analysis	varies with specific compounds e.g. Chlordane 0.5 mg/M^3	
Chlordecone (kepone)	•	•					Pleuritic and joint pains	•	•				Blood analysis	emergency standard 1 μg/M^3	1 μg/M^3
Estrogenic compounds															
Diethylstilbestrol					•			•	•			•	Blood analysis	not specified	not specified
Ionizing radiation (Whole body)															
X-rays and gamma rays				•			Gastro-intestinal disorders	•	•	•	•	•	Personal film badge dosimetry Thermoluminescent dosimetry	NCRP recommendation 1.25 rad/quarter 5 rad/year Pregnancy 0.5 rem/full pregnancy	
Miscellaneous substances															
Carbon disulfide	•				•		Retinopathy		•	•		•	Urine iodine-azide test	20 ppm	3 mg/M^3
Ethylene Oxide				•		•					•		CBC	50 ppm	50 ppm

• Animal and/or human data

Time weighted average 40-hr. week, 8-hr. day (OSHA), 10-hr. day (NIOSH).

In pregnancy, blood lead less than 40μg/100 gms is suggested

Evidence only male infertility, no data on females

SOURCE: National Institute for Occupational Safety and Health (9).

neoplastic respiratory disease were observed. The authors calculated the excess annual mortality due to lung cancer. When workers with low-to-moderate exposure experienced a mean excess lung cancer mortality of 63 deaths (per 100,000 years' exposure). Those severely exposed for less than 2 years experienced an excess of 44 deaths, and those severely exposed for 2 years or longer experienced an excess of 241 deaths. Interestingly, an examination of deaths did not reveal any significant association with age at first employment in the asbestos factory. In the sub-sample of workers whose smoking histories were available, those women who had both smoked and were heavily exposed had a risk of developing lung cancer over 30 times that of non-exposed nonsmoking women. The authors concluded that the data suggested that asbestos and cigarette smoking exert multiplicative rather than merely additive effects.

In summary, the data on smoking and asbestos exposure in women closely resemble the findings demonstrated for men.

COTTON DUST

Approximately 250,000 women were employed in the textile industry in 1978; that population included approximately 100,000 women engaged in spinning, twisting, and winding operations. Byssinosis is a syndrome characterized by tightness of the chest and shortness of breath in workers exposed to dust of cotton, flax, and hemp. In addition to these acute symptoms, workers have been found to develop chronic bronchitis, and some become severely disabled by their obstructive lung disease (3). Berry, et al. studied the workers in 14 cotton and 2 man-made fiber mills in England (1). They found that men had a greater prevalence of byssinosis than women, and that smokers of both sexes had 1.4 times greater prevalence of byssinosis than nonsmokers. Byssinosis prevalence was also positively associated with length of exposure to cotton dust in both women and men and was positively associated with dust level in the working environment in women. Berry, et al. were unable to determine if the observed difference in prevalence by sex represented a difference in physiologic response or differences in occupational exposure. They also found a higher prevalence of bronchitis in exposed versus nonexposed workers of both sexes. Smoking workers had higher bronchitis rates than nonsmoking workers.

Bouhuys, et al. studied 645 active and retired cotton textile workers (including 372 women), aged 45 and older, who had worked an average of 35 years. Their respiratory symptoms and flow-volume curves were compared to those of community resi-

TABLE 7.—Example of pulmonary irritants and inorganic sensitizers in various occupations where women work

Severe pulmonary irritant	Inorganic sensitizers	Occupation	# of women employed in thousands
Beryllium & Compounds Phosphorous Trichloride Tellurium (Hexaflouride) Zinc (Chloride fume)	Platinum Salts	Electronic Machinery, Equipment & Supplies	890
		—Household Appliances	67
		—Radio, T.V. & Communication Equipment	216
		—Electrical Machinery, Equipment & Supplies	604
Ammonia Chlorine Ozone Sulfuric Acid Uranium Compounds Vanadium Compounds (Pentoxide)	Phthalic Anhydride	Professional & Photographic Equipment & Watches	238
		—Scientific & Controlling Instruments	65
		—Optical & Health Services Supplies	119
		—Photographic Equipment & Supplies	36
Acrolein Ammonia Cadmium dust Chlorine Chromates Dichloroethyl ether Ethylene Oxide Hydrogen Chloride	Cobalt, metal fumes & dust Phthalic Anhydride	Rubber & Misc. Plastic Products	257
		—Rubber Products	86
		—Misc. Plastic Products	171

Severe pulmonary irritant	Inorganic sensitizers	Occupation	# of women employed in thousands
Hydrogen Fluoride			
Hydrogen Sulfide			
Phosgene			
Phosphorous Trichloride			
Phthalic Anhydride			
Sulfuric Acid			
Tellurium (Hexafluoride)			
Zinc Compounds			
Ammonia	Phthalic Anhydride	Leather & Leather Products	177
	Polyvinyl Chloride	—Footwear, except rubber	13
Chromic Acid & Chromates		—Leather Products, except footwear	40
Chromium, metals & insoluble salts			
Hydrogen Sulfide			
Phthalic Anhydride			
Sulphur Dioxide			
Ammonia		Fabricated Metal Products	299
Cadmium dust/fumes		—Cutlery, hand tools, & other hardware	52
Chromic Acid & Chromates		—Fabricated structural metal products	78
Chromium, metal & insoluble salts		—Screw machine products	26
Fluorine		—Metal stamping	43
Hydrogen Chloride		—Misc. fabricated metal products	101
Nitrogen Dioxide			
Sulfuric Acid			
Zinc Chloride fumes			

TABLE 7.—(Continued)

Severe pulmonary irritant	Inorganic sensitizers	Occupation	# of women employed in thousands
Chlorine Hydrogen Fluoride	Detergents (Enzymatic)	Personal Services —Laundering, Cleaning, & other Garment Services	231
Chlorine Chlorine Dioxide Chromium, metal & insoluble salts Nitric Acid Sulfur Dioxide Sulfuric Acid	Detergents (Enzymatic) Cobalt	—Beauticians	492
Ammonia		Private Households	1,217
Chlorine		Hotels & Motels	424
Beryllium & Beryllium compounds Chromic Acid & Chromates Chromium, metal & insoluble salts Iodine Selenium Hexafluoride Zinc Chloride fumes	Cobalt, metal fumes & dust Detergentes (Enzymatic) Platinum Salts	Professional & Related Services —Hospital Workers —Offices of Physicians —Offices of Dentists —Health Services —Convalescent Institutions	11,931 2,866 506 242 473 869

Severe pulmonary irritant	Inorganic sensitizers	Occupation	# of women employed in thousands
Ammonia	Cobalt dust	Textile Mill Products	409
Antimony	Phthalic Anhydride	—Knitting Mills	126
Bromine		—Yarn, thread & fabric mills	229
Cadmium dust fumes		—Misc. Textile mill products	23
Chlorine		Apparel & other fabricated	
Chromates		textile products	
Cotton dust, raw		—Apparel & Accessories	995
Dichloroethyl ether		—Misc. fabricated	898
Dimethylamine		textile products	97
Ethylene Chlorohydrin			
Ethylene Oxide			
Hydrogen Sulfide			
Methyl Bromide			
Nitric Acid			
Nitrogen Dioxide			
Sulfur Dioxide			
Sulfuric Acid			
Zinc Chloride fumes			
Hydrogen Sulfide		Meat Products	12,986

SOURCE: National Clearinghouse for Smoking and Health (8), Rones, P. (14), Stellman, J. (16).

dents who acted as controls (3). Textile workers of both sexes had significantly increased prevalence of chronic cough, wheezing, and dyspnea. Work in the textile mills was the major variable associated with symptom prevalence, with smoking as an additional significant variable. The lung function data confirmed the association of both smoking and working in the mills with decreased lung function. Non-smoking female workers were slightly more likely to report chronic cough than nonsmoking men, but smoking male workers were almost twice as likely to report this symptom as smoking women. A similar pattern was seen for wheezing and chest tightness, but not for dyspnea.

Kilburn, et al. studied the prevalence of byssinosis and bronchitis in 1,016 women textile workers and showed an interaction of smoking and work exposure in producing a higher prevalence rate of both byssinosis and bronchitis at a given dust level (5).

In summary, women have clearly been shown to have a higher risk of developing byssinosis, chronic bronchitis, and chronic obstructive lung disease because of exposure to cotton dust in the workplace. Cigarette smoking has been shown to interact with some work exposures to increase this risk, although it is not established whether this interaction is additive or multiplicative. Men employed in occupations where they are exposed to cotton dust have a greater prevalence of bronchitis and respiratory disability than women. Clarification is necessary to determine whether this is a sex difference or a difference in exposure (either occupational or smoking).

Summary

1. The 1979 Surgeon General's Report identified the ways in which smoking cigarettes may interact with the occupational environment. They include:

    a) Facilitation of absorption of physical contamination of cigarettes,

    b) Transformation of workplace chemicals into more toxic substances,

    c) Addition of the exposure to a toxic constituent of tobacco smoke to a concurrent exposure to the same constituent present in the workplace,

    d) Addition of a health effect due to environmental exposure to a similar health effect due to smoking,

    e) Synergy of exposures, and

    f) Causation of accidents.

2. Women are entering occupational environments with greater frequency, and thus may be experiencing greater exposures to physical and chemical agents.

3. Cohorts of women with a greater prevalence of smoking are currently reaching the ages of maximal disease occurrence, replacing earlier cohorts with lower cigarette exposures.

4. Physiologic differences in hormonal status between males and females constitute a potential source of differing responses.

5. In the workplace women who are pregnant present a 9-month exposure opportunity, including potential teratogenic and perinatal mortality effects.

6. Concurrent exposure of women to smoking and asbestos resulted in a clear excess of cancer of the lung.

7. Women smokers exposed to cotton dust run a higher risk of developing byssinosis, bronchitic syndromes, and abnormal pulmonary function tests than nonsmoking women.

References

(1) BERRY, G., MOLYNEUX, M.K.B., TOMBLESON, J.B.L. Relationships between dust level and byssinosis and bronchitis in Lancashire cotton mills. British Journal of Industrial Medicine 31: 18–27, 1974.

(2) BERRY, G., NEWHOUSE, M.L., TUROK, M.E. Combined effect of asbestos exposure and smoking on mortality from lung cancer in factory workers. Lancet 2(7775): 476–479, September 2, 1972.

(3) BOUHUYS, A., SCHOENBERG, J.B., BECK, G.J., SCHILLING, R.S.F. Epidemiology of chronic lung disease in a cotton mill community. Lung 154: 167–186, 1977.

(4) HAMMOND, E.C., SELIKOFF, I.J. Relation of cigarette smoking to risk of death or absestos-associated disease among insulation workers in the United States. In: Boguvski, P., Gilson, J.C., Timbrell, V., Wagner, J.C., Davis, W. (Editors). Biological Effects of Asbestos. International Agency for Research on Cancer, Scientific Publication No. 8, Lyon, France, International Agency for Research on Cancer, 1973, pp. 312–317.

(5) KILBURN, K.H., KILBURN, G.G., MERCHANT, J.A. Byssinosis: Matter from lint to lungs. American Journal of Nursing 73(11): 1952–1956, November 1973.

(6) NATIONAL CENTER FOR HEALTH STATISTICS. Health Interview Survey, 1976. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics. (Unpublished data)

(7) NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH. Adult Use of Tobacco, 1975. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, Bureau of Health Education, National Clearinghouse for Smoking and Health, June 1976.

(8) NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH. Survey of Health Professionals: Smoking and Health, 1975. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, Bureau of Health Education, National Clearinghouse for Smoking and Health, June 1976, 42 pp.

(9) NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH. Occupational Diseases (Revised Edition). Department of Health, Education, and Welfare, Public Health Service, Center for Dis-

ease Control, National Institute for Occupational Safety and Health, June 1977, 608 pp.

(10) NEWHOUSE, M.L. Cancer among workers in the asbestos textile industry. In: Buguvski, P., Gilson, T.C., Timbrell, V., Wagner, J.C., Davis, W. (Editors). Biological Effects of Asbestos. International Agency for Research on Cancer, Scientific Publication No. 8, Lyon, France, International Agency for Research on Cancer, 1973, pp. 203–208.

(11) NEWHOUSE, M.L., BERRY, G., WAGNER, J.C., TUROK, M.E. A study of the mortality of female asbestos workers. British Journal of Industrial Medicine 29: 134–141, 1972.

(12) POLLER, L., THOMSON, J.M., OTRIDGE, B.W., YEE, K.F., LOGAN, S.H.M. Effects of manufacturing oral contraceptives on blood clotting. British Medical Journal 1: 1761–1762, June 30, 1979.

(13) PROCTOR, N.H., HUGHES, J.P. Chemical Hazards of the Workplace. Philadelphia, J.B. Lippincott Company, 1978, 533 pp.

(14) RONES, P., LEON, C. Employment and unemployment during 1978: an analysis. Special Labor Force Report 218. Department of Labor, Bureau of Labor Statistics, 1979.

(15) SELIKOFF, I.J., HAMMOND, E.C., CHURG, J. Asbestos exposure, smoking, and neoplasia. Journal of the American Medical Association 204(2): 106–112, April 8, 1968.

(16) STELLMAN, J., DAUM, S.M. Work is Dangerous to Health. New York, Pantheon Books, 1973, 448 pp.

(17) U.S. DEPARTMENT OF LABOR. Employment and unemployment during 1978: An analysis. Department of Labor, Bureau of Labor Statistics, Special Labor Force Report 218, 1979.

(18) U.S. PUBLIC HEALTH SERVICE. Smoking and Health. A Report of the Surgeon General. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, DHEW Publication No. (PHS) 79-50066, 1979, 1251 pp.

PREGNANCY AND INFANT HEALTH

Introduction

A woman who smokes during pregnancy not only risks her own health, but also changes the conditions under which her baby develops. Studies have identified specific areas in which the effects of maternal smoking during pregnancy may occur. These include fetal growth, most often determined by comparing birth weights of smokers' babies with those of otherwise similar nonsmokers' babies; spontaneous abortions, fetal deaths, and neonatal deaths; pregnancy complications, including those that predispose to preterm delivery; possible effects on lactation; and long term effects on surviving children. The relationships between maternal smoking and these outcomes have been established by clinical, pathological, and especially epidemiological studies. Understanding of mechanisms by which smoking may produce the observed effects has been gained by physiological studies in humans and experimental studies in animals.

The Chapter on Pregnancy and Infant Health in the 1979 Surgeon General's Report is a detailed review of past studies of the effects of smoking in pregnancy, with a comprehensive bibliography. This section summarizes current knowledge in major areas of study, describes important new studies, and points out areas requiring further research (146).

Smoking, Birth Weight, and Fetal Growth

Babies born to women who smoke during pregnancy are, on the average, 200 grams lighter than babies born to comparable women who do not smoke. Since 1957, when Simpson reported this finding from her original study (138), it has been confirmed in more than 45 studies of more than half a million births (146). Results of these studies are expressed as mean birth weights of smokers' and nonsmokers' babies or, alternatively, as the percentage of babies who weigh less than a specified amount, usually 2,500 grams.

To illustrate the association between maternal smoking and an increased proportion of low-birth-weight infants, the results of five studies with an aggregated total of almost 113,000 births in Wales, the United States, and Canada are summarized in Table 1. In these populations, 34 to 54 percent of the mothers smoked during pregnancy and on the average the smokers had twice as many low-birth-weight babies as the nonsmokers. Also in these populations, from 21 to 39 percent of the incidence of

TABLE 1.—Birth weight under 2,500 grams by maternal smoking habit, relative and attributable risks derived from published studies

Study	Nonsmokers No.	Smokers No.	Smokers Proportion	Births < 2,500 grams Nonsmoker (%)	Births < 2,500 grams Smoker (%)	Relative risk smoker: nonsmoker	Attributable risk* (%)
Cardiff	7,176	6,238	.465	4.1	8.1	1.98	31
US Collaborative							
White	8,466	9,781	.536	4.3	9.5	2.21	39
Black	11,252	7,777	.409	10.7	17.5	1.64	21
California, Kaiser Permanente							
White	3,189	2,145	.402	3.5	6.4	1.83	25
Black	934	479	.338	6.4	13.4	2.09	27
Montreal	3,954	3,004	.432	5.2	11.4	2.19	34
Ontario	27,316	21,062	.435	4.5	9.1	2.02	31

*Percentage of total birth weights <2,500 gm attributable to maternal smoking. Attributable risk in population = b(r−1) divided by b(r−1) +1 where b = proportion of mothers who smoke and r = relative risk of low weight = smoker rate/nonsmoker rate.

SOURCE: Meyer, M.B. (86).

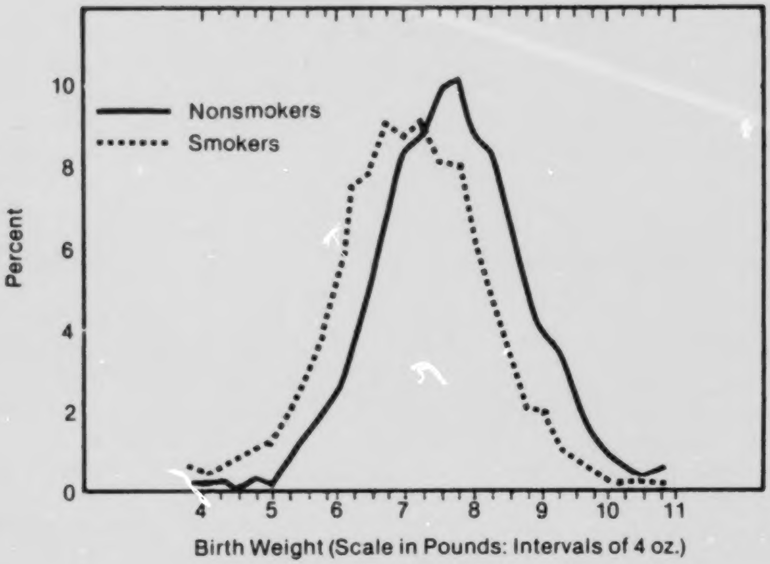

FIGURE 1.—Percentage distribution by birth weight of infants of mothers who did not smoke during pregnancy and of those who smoked one pack or more of cigarettes per day

SOURCE: MacMahon, B. (77).

low birth weight could be attributed to maternal smoking (3,15,38,86,102,106,107).

One study in which rates of low birth weight were simultaneously adjusted for multiple factors showed that maternal smoking had a more significant relationship to birth weight than did previous pregnancy history, hospital pay status, mother's prepregnant weight, height, age-parity, or sex of child. Adjusted rates of birth weights under 2,500 grams were 49 per thousand for nonsmokers, 76 per thousand for smokers of less than a pack per day, and 114 per thousand for smokers of a pack per day or more. The risk of having a low-birth-weight baby therefore increased 53 percent and 130 percent for light and heavy smokers, respectively, compared with nonsmokers (86).

Population studies that illustrate whole distributions of birth weights by maternal smoking levels show a downward shift of all birth weights in proportion to the amount smoked (74, 77,83,114,136,160) (see Figure 1).

These studies show that the relationship between smoking and reduced birth weight is independent of all other factors that influence birth weight, such as race, parity, maternal size, socioeconomic status, sex of child, and other factors that have been studied. It is also independent of gestational age. There is a dose-response relationship: that is, the more the woman smokes during pregnancy, the greater the reduction in birth weight. If a woman gives up smoking by her fourth month of gestation her risk of delivering a low-birth-weight baby is similar to that of a nonsmoker.

PLACENTAL RATIOS

Analyses of placental weights by maternal smoking habits have noted that these weights were either not affected or were less affected by maternal smoking than were birth weights (57,61,91,104,155). The placental ratio, the ratio of placental weight to birth weight, tended to be larger for smokers than for nonsmokers, mainly because of the dose-related reduction in birth weights with increasing number of cigarettes smoked.

Wingerd and colleagues have studied placental ratios based on data from 7,000 pregnancies among members of the Kaiser Foundation Health Plan in Oakland, California (156). Smoking information was obtained early in pregnancy, and placentas were handled according to Benirschke's standardized protocol. Figure 2 shows placental ratios by smoking level and gestation for single live births. At each gestational age, from 37 through 43 weeks, the more the mother smoked during pregnancy, the higher was the placental ratio. These ratios were higher for black than for white women and tended to increase as maternal hemoglobin level decreased (156).

Christianson's recent report, based on standardized examinations of these placentas, has shown that the increase in placental ratio with maternal smoking level was due to considerable decreases in mean birth weight, accompanied by slight increases in mean placental weight. In addition, smokers' placentas were significantly thinner than those of nonsmokers, and their minimum diameters were larger (19).

Maternal smoking leads to significant increases in carboxyhemoglobin in maternal and fetal blood, with a consequent reduction in the oxygen carrying capacity of both, and a reduction of the pressure at which oxygen is delivered to the fetal tissues (70,72,146). Christianson discusses the similarity between studies of placental ratios by smoking level, altitude, maternal anemia, and maternal cyanotic heart disease. She suggests that the changes in placental ratio represent an adap-

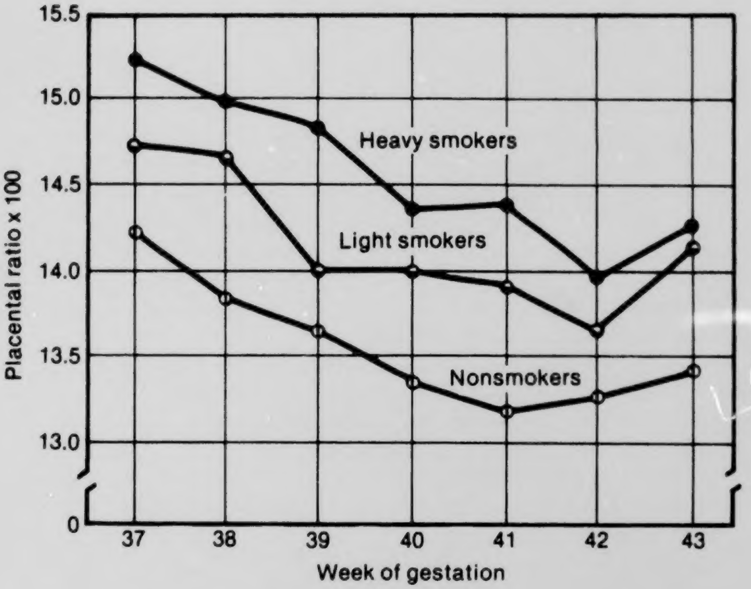

FIGURE 2.—Ratio of placental weight to birth weight by length of gestation and maternal smoking category

SOURCE: Wingerd, J. (156).

tation to relative fetal hypoxia (19). An adaptive advantage for survival might occur because a larger placenta with an increased area of attachment would deliver more oxygen, and a smaller fetus would have a decreased oxygen demand. If so, it is extremely important to know whether this reduction in size is accompanied by any long-term costs in later growth and development.

GESTATION AND FETAL GROWTH

In early studies the consistent finding that mean birth weights were lower and the frequency of births under 2,500 grams higher for women who smoked during pregnancy than for similar nonsmokers raised the obvious question of whether this might be due to a smoking-related reduction in gestation. This is not the case. Studies consistently show that mean gestation is minimally reduced by maternal smoking (less than 2 days) (3,13,146,159) and that birth weight is lower for infants of smokers than for infants of nonsmokers at each gestational age (3,15,83,146).

The finding that maternal smoking does not cause an overall downward shift in the distribution of gestational ages, as was shown for birth weights of smokers' infants, leads to the conclusion that the lower weight must be due to direct retardation of fetal growth. In other words, these infants are small-for-dates rather than preterm. The type of fetal growth retardation associated with maternal smoking is characterized by an abnormally short crown–heel length for gestational age (89,90). Smokers' babies are smaller than corresponding nonsmokers' babies in all dimensions measured, including length, head circumference, chest circumference, and shoulder circumference (10,30,31,52,57,61,102,104,146,157).

Previous studies of these measurements at birth have inferred that birth size reflects the rate of fetal growth; this has been confirmed by a definitive study in which fetal biparietal diameters were measured serially during gestation. Persson and coworkers studied 5,715 pregnancies prospectively, making ultrasonic measurements of biparietal diameters (BPD) from 18 to 20 weeks through term. Separate growth curves of BPD were constructed for fetuses of smokers and nonsmokers who were delivered between 266 and 294 days after the last menstrual period. The BPD increased faster in the nonsmoking group; the difference from the smoking group was significantly apparent from the 28th week and was positively correlated with the average number of cigarettes smoked (Figure 3). Measurements taken at birth showed that the distributions of birth weights and lengths shifted downwards in proportion to the level of smoking. Figure 4 illustrates this shift (114). These findings corroborate Miller's characterization of smokers' babies as normally proportioned but short as well as light for dates, and smaller in all dimensions than babies of nonsmokers (90). The data are also consistent with the speculation that relative fetal hypoxia results in a slower mitotic rate, a baby with fewer cells, and a reduced oxygen demand.

LONG-TERM GROWTH AND DEVELOPMENT

Possible long-term consequences of maternal smoking during pregnancy are also of concern. Several long-term studies provide evidence that children of smoking mothers have slight but measurable deficiencies in physical growth, intellectual and emotional development, and behavior (95).

Because these complex outcomes are affected by many known and unknown factors, it is important to take these other factors into account in any attempt to measure long-term effects of maternal smoking. Several well-controlled studies have shown

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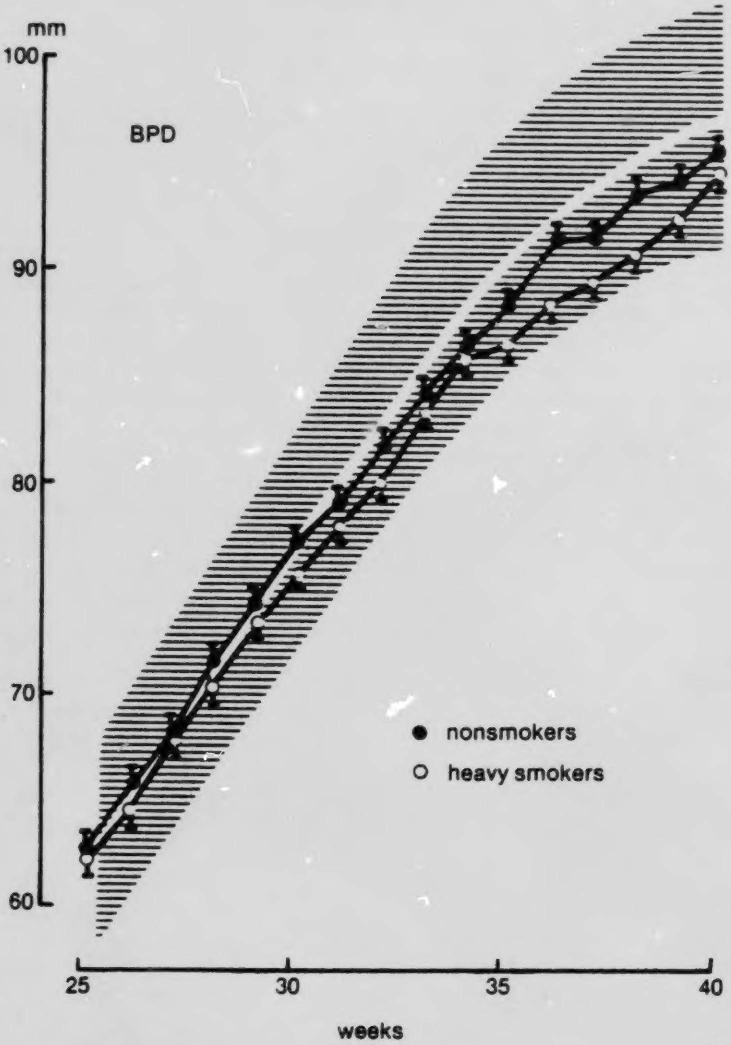


FIGURE 3.—Fetal Biparietal Diameters (BPD) values [means and standard error of means (SEM)] of nonsmokers and heavy smokers (10 cigarettes/day) plotted in relation to postmenstrual age against the normal range (shaded area depicts 95% confidence interval)

SOURCE: Persson, P.H. (114).

that the physical growth of smokers' babies remains behind that of nonsmokers' babies as measured at 7 to 14 days (31): 1 year, 4 years, and 7 years (pairs of births matched for race, date of delivery, maternal age and education, and sex of child) (52); 5 years (adjusted for other factors) (157); up to 6½ years (prospective study) (35); and at ages 7 and 11 (follow-up studies of the 17,000 children from the British Perinatal Mortality Study, with the adjustment for other social and biological factors) (16,30,33).

Associations have also been noted between maternal smoking and deficiencies in neurological and intellectual development of the child. Hardy and Mellits analyzed findings for 88 pairs of children of smokers and nonsmokers, matched for race, date of delivery, maternal age and education, and sex of the child. Although they reported no significant differences in intellectual function between children born to smoking and nonsmoking mothers, the direction of difference on almost all tests was in favor of the nonsmokers' babies. Fewer smokers' than nonsmokers' children had normal neurological status at age 1 year, both in the original 88 matched pairs and in the additional set of 55 pairs of children of smokers and nonsmokers, matched for birth weight as well as for the other cited factors. In both sets, smokers' children had lower scores on the majority of tests of intelligence and intellectual function at ages 4 and 7 (52,146).

Similarly, Dunn evaluated neurological, intellectual, and behavioral status in a prospective study of low-birth-weight infants, including 76 who were "small-for-dates" (term and preterm), 92 "truly premature" (preterm with birth weight between 11 and 89 percentile) and 151 full-birth-weight control infants. Neurological abnormalities, including minimal cerebral dysfunction and abnormal or borderline electroencephalograms, were slightly more common among children born to women who smoked (Table 2).

In a battery of psychological tests, the mean scores of children of nonsmoking mothers were better than those of smokers' children in 45 out of 48 correlations, and the difference was significant in 14 of these. Some significant differences in favor of nonsmokers' children were also demonstrated with respect to behavior ratings and school placement (35). These results are very similar to those of Hardy and Mellits in that the direction of the differences was almost always in favor of the nonsmoker's child.

Small numbers and population selection factors were not a problem in the longitudinal follow-up of the population originally included in the British Perinatal Mortality Study, comprising approximately 17,000 births, an estimated 98 percent of all births in England, Scotland, and Wales during the week of

TABLE 2.—Incidence of neurological abnormalities at about 6½ years, by maternal smoking habits

Diagnosis	Percent of Children with Diagnosis Maternal Smoking Habits Smoker	Nonsmoker	P
Minimal cerebral dysfunction	20.0	11.0	<.05
Total neurological abnormalities	29.4	19.5	<.05
EEG borderline or abnormal			
Low-birth-weight children	46.3	32.4	NS
Full-birth-weight children	28.2	21.6	NS

NS = not significant.
SOURCE: Dunn, H.G. (35).

March 3 to 9, 1958. These children have been traced and studied again at ages 7 and 11, to describe their behavior, their health, their physical development, their educational standards, and their home environment. At ages 7 and 11 years, physical and mental problems due to maternal smoking during pregnancy were found, and these increased with the number of cigarettes smoked.

Children whose mothers smoked 10 or more cigarettes a day during pregnancy were on average 1.0 centimeter shorter and 3 to 5 months retarded in reading, mathematics, and general ability, as compared with the offspring of nonsmokers. After allowing for associated social and biological factors, all of these differences were highly significant, as illustrated in Figure 5 ($p<0.001$) (16,30).

Denson's case-control study of hyperkinesis reported a highly significant association of hyperkinesis with heavy maternal smoking, which at a mean level of 23.3 cigarettes per day was more than three times the average for two control groups. The authors concluded that their findings were "consistent with the hypothesis that smoking during pregnancy is an important cause of the hyperkinetic syndrome" (31).

A recent comparison by Saxton of behavioral patterns of infants of mothers who smoked during pregnancy with infants of mothers who did not smoke found that these patterns can be influenced by smoking in pregnancy, and that the auditory senses are particularly affected. Fifteen smokers of more than 15 cigarettes per day and 17 nonsmokers were selected for study, matched for maternal age, social class, and parity. All infants were spontaneous term deliveries of normal birth weight. Sex distribution, length of labor, analgesia, and obstetrical factors were similar for the two groups. Examiners who did

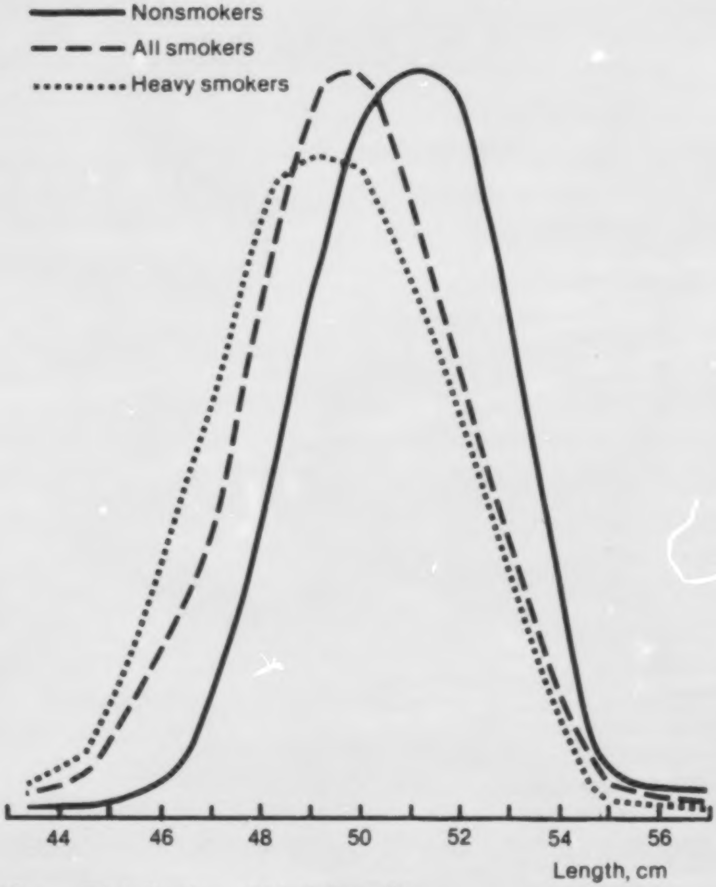
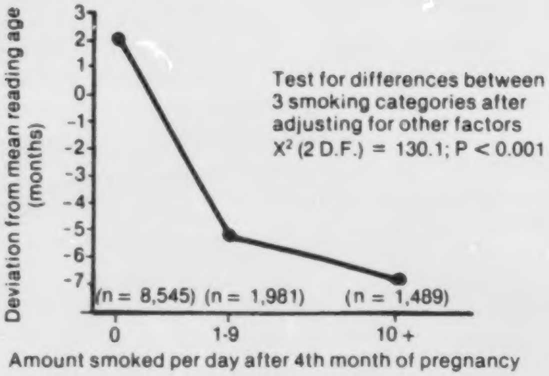


FIGURE 4.—Distribution of birth lengths

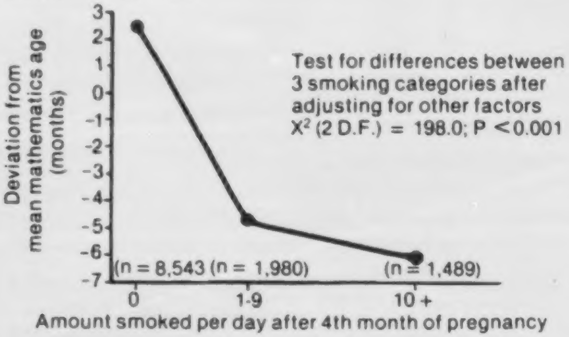
SOURCE: Persson, P.H. (114).

not know the smoking status of the mother evaluated the infants at 4 to 6 days of age, using the Brazelton Neonatal Behavioral Assessment Scale. The scale includes a total of 20 tests and maneuvers. While many of these showed no statistically significant differences, auditory tests or tests with auditory components were significantly different. Recorded "overall impressions" of the infants at the end of the test showed that the smokers' infants tended towards "irritability, decreased ability for self-control, and a general lack of interest, whereas the nonsmokers, infants tended to be less irritable and better oriented." The author concluded that some effect on the normal

a) Reading Comprehension



b) Mathematics Ability



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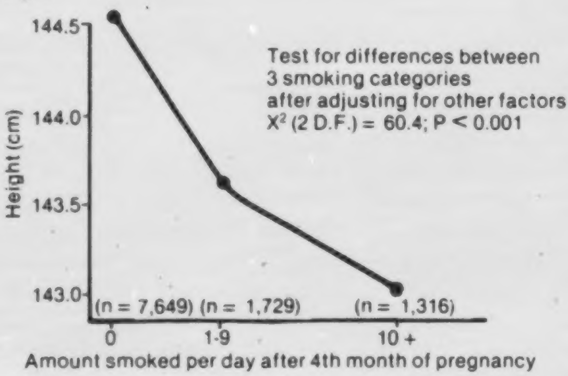

FIGURE 5.—Tests of 11-year-old children by mothers' smoking habits after the fourth month of pregnancy

SOURCE: Butler, N.R. (16).

hearing mechanism had occurred in infants of smokers, possibly due to a hypoxic effect of carbon monoxide on the cochlear organ during development (132).

These studies suggest unfavorable effects of maternal smoking during pregnancy on the child's long-term growth, intellectual development, and behavioral characteristics. Although these changes are difficult to study because of the vast complexity of possible antecedent and confounding variables, high priority should be given to obtaining conclusive answers about the long-term consequences of fetal exposure to cigarette smoke.

ROLE OF MATERNAL WEIGHT GAIN

In the search for mechanisms through which maternal smoking reduces birth weight, the question has been asked whether it might be an indirect result of reduced appetite, less intake of food, and lower maternal weight gain (84,127). Several early studies reported no differences between smoking and nonsmoking women in intake of food or in weight gain, and concluded that the effect of maternal smoking on birth weight was not mediated in this way (146).

Meyer analyzed the relationships between maternal smoking, birth weight, maternal weight gain, and gestation, using data based on 31,788 births from the Ontario Perinatal Mortality Study (106,107). She found a significant downward shift in the distribution of birth weights as maternal smoking level increased, but no similar shift in the distribution of maternal weight gain with smoking. Whereas the usual strong relationship between the proportion of births under 2,500 grams and maternal smoking level was found, there was no similar trend for the proportion of mothers who gained less than 10 pounds during pregnancy. Finally, the proportion of infants weighing less than 2,500 grams increased directly with the amount smoked within each maternal weight gain group from less than 5 pounds to 40 pounds or more, as shown in Figure 6 (83). From Figure 6, one might conclude that smoking has a more pronounced effect on low birth weight when maternal weight gain during pregnancy is less than 20 pounds.

Other studies have indicated a lack of relationship between smoking and maternal weight gain, while demonstrating a direct relationship between smoking and fetal growth rate. The German prospective study of 6,200 pregnant women, examined every month from the first trimester through delivery, showed no significant association between smoking habit and weight gain. The usual relationships were found between smoking and

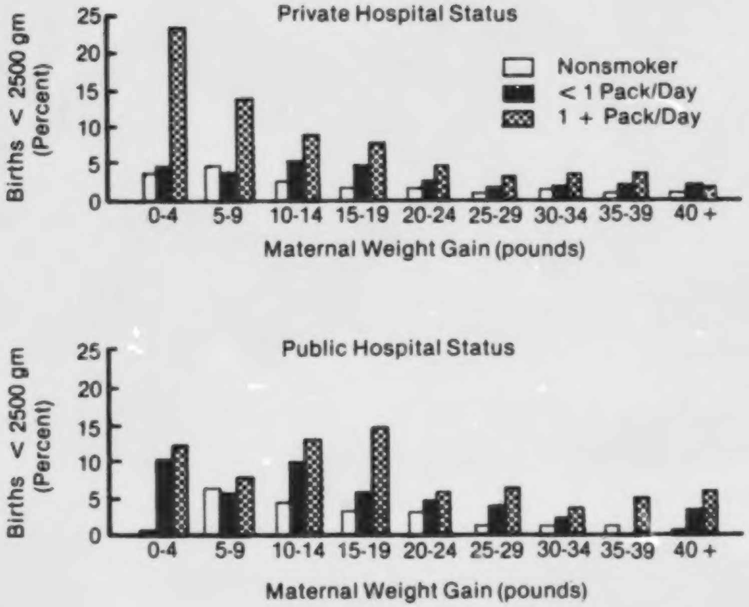

FIGURE 6.—Percentage of birth weights under 2,500 grams by maternal smoking level within maternal weight gain group (five-pound intervals) by hospital pay status. Births of 38 + weeks gestation (Ontario study)

SOURCE: Meyer, M.B. (83).

small-for-dates babies, with general retardation of weight, length, and head circumference in proportion to the number of cigarettes smoked during pregnancy (80). Miller and Hassanein also found that the effects of smoking on fetal growth did not appear to be related to maternal nutrition (93). Persson's study showing retardation of fetal growth of smokers' babies by serial measurement of biparietal diameters and by weight, length, and other measurements at birth showed that the low birth weights were independent of maternal weight gain. These authors concluded that the fetal growth retardation resulted from a direct pharmacological effect of smoking on the fetus "rather than an influence resulting from nutritional deprivation" (114).

Hajeri and colleagues studied maternal weight gain in 105 smokers of 10 or more cigarettes a day with a control group of nonsmokers who were similar with respect to gestation, age, height, parity, and maternal weight at conception. Birth weights, specific for sex, were significantly higher for infants of

TABLE 3.—**Birth weight under 2,500 gm by maternal smoking and prepregnant weight**

		Births <2,500 gm per 100 Total Births Maternal Smoking (Packs per day)			Ratio Smoker:Nonsmoker	
Prepregnant Weight	Total Births	0	<1	1+	<1	1+
<120 lb (<54 kg)	18,935	6.1	10.2	15.8	1.7	2.6
120–134 lb (54–61 kg)	19,798	4.2	6.3	9.5	1.5	2.3
135+ lb (>61 kg)	10,456	3.3	5.1	8.7	1.6	2.6

SOURCE: Meyer, M.B. (86).

nonsmokers, with a mean difference for boys of 330 grams and for girls of 320 grams ($p<.01$). Mean extrauteral weight gain, calculated as the difference between maternal weight gain and the weights of fetus and placenta, was 7,044 grams for smokers and 6,899 grams for nonsmokers (49).

Garn has compared mean birth weights, specific for gestational age, of babies of obese smokers, all nonsmokers, and all smokers, using data from the Collaborative Perinatal Project of the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS). Obesity was defined as the top 15 percent of the distribution of prepregnant weights, shown separately for black and white women. Babies of the 1,383 obese white smokers had mean birth weights similar to the total group of white nonsmokers and higher than the total group of white smokers. The 1,001 obese black smoking mothers had babies whose mean birth weights were generally higher than those of all black nonsmokers, leading Garn to conclude that "maternal obesity (weight-defined) apparently counteracts the smoking effect on the conceptus" (43). Because birth weight is strongly correlated with maternal size, a more appropriate comparison would have been between mean birth weights of the babies of obese smokers and the babies of obese nonsmokers. That such a comparison would show the usual relationship to maternal smoking level is suggested by Meyer's analysis of birth weight by maternal smoking and prepregnancy weight (Table 3). The correlation between maternal weight and the proportion of low-birth-weight babies is clear at each smoking level, and the independent relationship between smoking level and low birth weight is clear at each level of maternal weight.

TABLE 4.—Mean birth weights in successive pregnancies to the same women, by smoking habit

Smoking Habits First pregnancy	Smoking Habits Second pregnancy		Mean Birth Weight (gm)		
		N	# 1	# 2	Difference 2nd–1st (gm)
Smoker	Smoker	886	3204	3228	+24
Nonsmoker	Nonsmoker	988	3356	3388	+32
Difference: Nonsmoker - Smoker (gm)			+152	+160	
Smoker	Nonsmoker	119	3271	3381	+110
Nonsmoker	Smoker	108	3323	3265	–58
Difference: Nonsmoker - Smoker (gm)			+52	+116	

SOURCE: Naeye, R. (93).

The relative increases in the proportion of low-weight births with light and with heavy smoking are almost identical in the three strata of prepregnant weight (86).

Studies of birth weight, maternal weight, and maternal weight gain should also be carefully controlled for maternal age and parity. In studies of successive births to the same mother included in the Collaborative Perinatal Project of the NINCDS, Garn found that prepregnancy weights increased with successive pregnancies by similar amounts for smokers and nonsmokers (44). Naeye, using the same data base, reported that maternal weight gain was less in the second pregnancy than in the first pregnancy for smokers, for nonsmokers, and for women who changed habits between pregnancies in either direction (93). Second babies weighed on the average 24 grams more than first babies if the mother smoked both times, and 32 grams more if the mother smoked neither time (Table 4). If the mother smoked during the first and not during the second pregnancy, the second baby weighed an average of 110 grams more than the first baby; in women who smoked during the second pregnancy but not during the first pregnancy, second babies averaged 58 grams *less* than first babies (93).

The most careful analyses indicate that the effect of maternal smoking is a direct one not mediated through an effect on maternal appetite, eating, or weight gain. In conclusion, as stated in a Lancet editorial, "the appeal of the nutritional hypothesis is that women might be more readily encouraged to eat more during pregnancy than discouraged from smoking. . . . However, if, as now seems more likely, the growth-retarding effect of smoking is due to fetal hypoxia, there is no short-cut to removing this adverse influence" (63). This conclusion in no way obviates the enormous importance of dietary factors during pregnancy.

Overt maternal malnutrition is associated with inadequate growth. Recently, it has been suggested that more subtle alterations in the maternal supply of essential nutrients combined with compromised uteroplacental circulation may contribute to reduced fetal growth. Crosby, et al. (26) observed that the concentrations of each of 14 amino acids and carotene were reduced significantly in the blood of smoking mothers. These workers postulated that, while these differences were on the order of 10 or 20 percent, they could be an important factor in producing the small-for-gestational-age infants associated with maternal smoking. In a study of over 1,100 pregnant women, Schorah, et al. (135) noted an inverse correlation between the number of cigarettes smoked and the leukocyte ascorbic acid concentration. For instance, the leukocyte ascorbic acid concentration was about 22 percent less in the blood of women who smoked more than 20 cigarettes a day as compared with controls. Despite a 15 percent increase in the number of circulating leukocytes in the blood of smokers, the blood ascorbic acid concentration was still 10 percent less than in controls. These differences were even more marked in women from lower socioeconomic groups. The authors suggested that in addition to the role of ascorbic acid in fetal nutrition, these lowered concentrations might be related to the increased incidence of premature rupture of the amniotic membranes in smoking women.

Smoking, Fetal and Infant Mortality, and Morbidity

SPONTANEOUS ABORTION

Past studies have demonstrated a statistically significant association between maternal cigarette smoking and spontaneous abortion (55,61,104), some showing a strong dose–response relationship (110,144,162). Spontaneous abortions are difficult to study because of problems of ascertainment. In prospective studies, early abortions may be missed, and bias may occur if one group tends to register earlier than the other. Retrospective studies allow more complete ascertainment but are subject to errors of recall. Nevertheless, higher rates of spontaneous abortion have been associated with maternal smoking in both types of studies (61,104,162).

Kullander and Kallen found higher rates of "spontaneous abortion" among smoking women, but noted that many of these pregnancies were unwanted. Analysis of their data showed that the relative risk of spontaneous abortion of smokers compared with nonsmokers was 1.20 for wanted and 1.35 for unwanted pregnancies (61). A case–control study of spontaneous abortion with important variables held constant reported an 80 percent increase in the odds of smoking among the cases compared with controls (60).

Recent studies corroborated the finding of associations between smoking and spontaneous abortion risk. In a small retrospective study in New Zealand, Fergusson found that women who smoked more than 20 cigarettes a day had almost twice the nonsmoker risk of having had a previous spontaneous abortion, and that the association could not be explained by differences in maternal age, educational level, parity, race, socioeconomic status or marital status (42). In a study of 12,013 consecutive pregnancies in Dublin, Ireland, Murphy and Mulcahy found a positive association between the number of cigarettes smoked and the rates of spontaneous abortion, independent of the effects of maternal age and parity. The authors stated that induced abortions are a negligible factor in Ireland and concluded that maternal smoking leads to reduced reproductive efficiency at all stages of pregnancy (92). Himmelberger and colleagues surveyed a group of professional women in medicine concerning the influence of maternal smoking on their 12,194 pregnancies (54). After controlling for interfering variables, the risk of spontaneous abortion for certain subgroups of heavy smokers was estimated to be as much as 1.7 times that for nonsmokers. Spontaneous abortion rates were lowest in the 25 to 29 year old category, increasing with age to levels of 33 and 36 percent for nonsmokers and smokers, respectively, at age 40 plus. The relative increase in risk associated with maternal smoking was highest at the youngest ages and decreased with increasing age (54).

An editorial in the British Medical Journal summarized these findings and stated: "Cigarette smoking, one of the first manifestations of women's social emancipation, is emerging as a possible threat to her procreative role." The proportion of abnormal karyotypes in abortuses of women who smoke appears to be reduced rather than increased (1). The mechanism underlying the smoking-related excess appears to be due to complications of pregnancy rather than to any fetal abnormality (13).

CONGENITAL MALFORMATIONS

Several studies have reported perinatal, fetal, or neonatal mortality rates by cause. In these comparisons, death rates due

TABLE 5.—Incidence of congenital abnormality (all single births)

	Nonsmokers		Smokers	
	Number	Percent	Number	Percent
Total abnormal infants		2.37		2.73
Type of abnormality				
Anencephaly	18	0.2	15	0.2
Spina bifida	20	0.22	23	0.3
Other C.N.S. abnormality	38	0.42	36	0.47
Cardiovascular abnormality	34	0.37	32	0.42
Gut abnormality	21	0.23	24	0.32
Genito-urinary abnormality	39	0.43	25	0.33
Bone abnormality	65	0.72	52	0.68
Cleft palate and/or hare lip	10	0.11	20	0.26
Other abnormality	19	0.21	18	0.24

x^2 (all abnormalities) = 2.22, $p > 0.05$.

x^2 (cleft palate and hare lip) = 5.36, $0.01 < p < 0.05$.

SOURCE: Andrews, J. (3).

to congenital malformations have usually been lower for smokers' than for nonsmokers' infants (3,22,46,87). This is compatible with the finding that smoking-related spontaneous abortions have a lower frequency of abnormal karyotypes and tend to occur later than spontaneous abortions in nonsmokers. As previously described, increased losses of conceptus associated with maternal smoking appear to be due to pregnancy problems and complications rather than to abnormalities of the embryo or fetus (41). Andrews and McGarry, in a community study of 18,631 pregnancies in Cardiff, Wales, reported that smokers' infants had lower mortality rates from malformations than those of nonsmokers. Rates of stillbirths due to congenital malformations were 0.32 and 0.27 per 100 nonsmokers and smokers respectively. Corresponding rates for neonatal deaths were 0.33 and 0.31 per 100 babies of nonsmoking and smoking mothers. On the other hand, the incidence of congenital malformations among all single births in Andrews' population was higher among smokers' babies, overall, and specifically higher for cleft palate and lip. Among other sites, some were higher for smokers and some for nonsmokers, as is shown in Table 5 (3).

A significant positive association between cardiac malformations and maternal smoking was shown by Fedrick and colleagues, based on firm diagnoses among stillbirths, neonatal deaths, and survivors to age 7 from the British Perinatal Mortality Survey. However, this difference was largely due to the inclusion of patent ductus arteriosus, which may or may not be classified as a malformation (80).

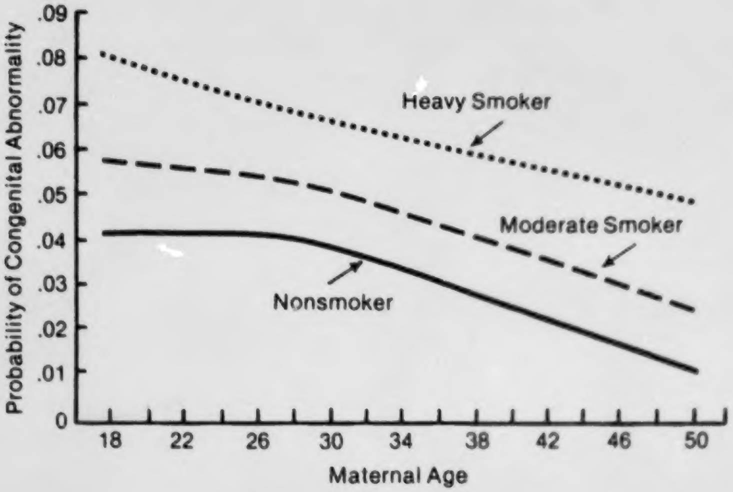

FIGURE 7.—Risk of congenital abnormality according to age and smoking habit

SOURCE: Himmelberger, D.U. (54).

Some recent studies have shown a positive association between maternal smoking and congenital malformations, defined in a variety of ways. Himmelberger and colleagues carried out a mail survey of professional women in medicine (54). They were interested in exposure to anesthetic gases in the operating room, and evaluated possible effects on pregnancy outcome of a number of factors including cigarette smoking. Information was obtained and analyzed by a multiple logistic regression based on 12,914 pregnancies, including 10,523 live births, which represented a response rate of 53.2 percent. After the effects of age, exposure to anesthetic gases, and pregnancy history were controlled, the risk of congenital abnormalities for babies of mothers who smoke was estimated. A statistically significant risk ($p < .05$) for maternal smoking was found. Figure 7 shows the estimated risk of congenital abnormality as a function of maternal age for nonsmokers, moderate smokers (1 to 19 per day), and heavy smokers (20 plus per day). Relative risks for heavy smokers compared with nonsmokers were as high as 2.3. Rates of abnormalities in each general category were higher for the children of smokers (see Table 6). The significant increase in cardiovascular abnormalities among smokers' children is in agreement with Fedrick's findings (40) and in general agree-

TABLE 6.—Comparison of congenital abnormality rates for babies born of smokers and nonsmokers, by type of abnormality

	Smokers		Nonsmokers		p*
Abnormality	%	No.	%	No.	
Cardiovascular	19.07+	(68)	13.65	(95)	0.02
Respiratory	15.15	(54)	12.07	(84)	0.10
Musculoskeletal	23.84	(85)	19.69	(137)	0.08
Gastrointestinal	13.46	(48)	9.48	(66)	0.04
Central nervous system	11.50	(41)	10.20	(71)	0.27
Urogenital	21.32	(76)	15.81	(110)	0.02

*One-tail significance level for the test of the difference between two proportions.

+Rate is number of congenital abnormalities per 1,000 live births. Rates based upon 3,565 live births among the smokers and 6,958 live births among the nonsmokers.

SOURCE: Himmelberger, D.U. (54).

ment with the study of Andrews and McGarry (3). Himmelberger, et al. point out that their findings are based on retrospective survey data, obtained by mail, and therefore subject to bias from various sources, including that of a high nonresponse rate. However, the study methods have been designed to eliminate those effects (54).

A recent study by Borlee and Lechat controlled for confounding variables by matching births with congenital malformations to control births according to hospital and time of birth, maternal age, sex of child, and socioeconomic level of parents. Two hundred and two children with malformations diagnosed at birth were compared with 175 controls, from a total of 17,970 consecutive births studied from June 1972 through May 1974. No differences were found between cases and controls in the distribution of smoking habits, including the number of cigarettes smoked with or without filters. Sixty-six percent of mothers of malformed infants and 68 percent of mothers of controls were nonsmokers. Fathers' smoking habits were also similar among cases and controls. Significantly more mothers of malformed infants were heavy coffee drinkers (8 plus cups per day). Because of the frequent association between heavy coffee drinking and smoking, both habits should be included in studies of environmental factors possibly related to the risk of congenital malformations (10). The same is true for consumption of alcohol in populations where drinking is prevalent.

Mau and Netter have reported births by gestation, birth weight, perinatal mortality, and the incidence of congenital malformations by smoking habits of fathers in 3,696 cases in which the mother was a nonsmoker. Trends toward lower birth weights and more preterm births with increasing levels of paternal smoking were not statistically significant. In the total study of 5,200 births, regardless of maternal smoking habits, there was a significant increase in the incidence of severe malformations with increasing levels of paternal smoking; children of heavily smoking fathers had about twice the expected incidence. Although malformations in all systems were more frequent if the father smoked over 10 cigarettes per day, only the differences in facial malformations were significantly different ($p<.01$) by smoking level. The authors state that the trends with paternal smoking were independent of maternal smoking level, maternal and paternal age, and social class (120).

More studies of these possible relationships are urgently needed. As serious malformations are relatively rare, the case–control approach is probably the method of choice, with careful matching of cases with suitable controls.

PERINATAL MORTALITY

The 1973 report, The Health Consequences of Smoking and the 1979 Report have summarized studies demonstrating a direct relationship between level of maternal smoking and risk of perinatal loss. The reports have also clarified reasons for the variation in risk observed in these studies (146,147).

Two important reasons for variability between studies have been demonstrated. First, other important variables such as age, parity, race, and socioeconomic status influence the results if they are unequally distributed between comparison groups of smokers and nonsmokers (89). Second, cigarette smoking is more harmful to the pregnancies of certain women than to those of others. In general, women with other risk factors were at greater risk from smoking than otherwise low-risk women (3,15,22,128,144,159).

Table 7 illustrates these points. It shows that women characterized by low social class, low level of education, being very young or old during pregnancy, or being black, have higher risks of perinatal mortality than their counterparts. Their increase in risk due to smoking is relatively greater. Meyer, et al. measured the perinatal mortality risks of light smokers (less than a pack of cigarettes per day) and of heavy smokers (one pack or more per day) relative to nonsmoker risks within subgroups of the population. The increased risk of perinatal mortality for light smokers who were young, low-parity, and nonanemic was less than 10 percent. At the other extreme, mothers characterized by high-parity, public hospital status, previous

TABLE 7.—Examples of perinatal mortality by maternal smoking status related to other subgroup characteristics

Study Population	No. of births		Category	Perinatal or neonatal deaths/1,000 births		Relative risk*
	Non-Smokers	Smokers		Non-smokers	Smokers	
British Perinatal Mortality Survey, England, all births	11,145	4,660	Social class			
			1,2 (high)	25.8	26.3	1.02
			3-5	33.5	46.6	1.39
Washington Co. Maryland, white	7,646	4,641	Father's education			
			9+ years	14.4†	16.1†	1.12
			≤ 8 years	17.6†	38.0†	2.16
Northern Finland, white	8,898	2,346		23.2	23.4	1.01
California, middle to upper middle class			Race			
	6,067	3,726	White	11.0†	11.3†	1.03
	2,219	1,071	Black	17.1†	21.5†	1.26
Boston City Hospital Prenatal Clinic			Race			
	513	892	White	29.2	31.4	1.08
	1,225	636	Black	28.6	54.1	1.89
Quebec, 10% sample of registered births	3,912	2,967	Maternal age			
			<25	12.1	16.1	1.33
			25-34	12.6	13.2	1.05
			35+	23.0	41.7	1.81

*Ratio of mortality rate for smokers' to nonsmokers' babies.

†Neonatal only.

SOURCE: Meyer, M.B. (88).

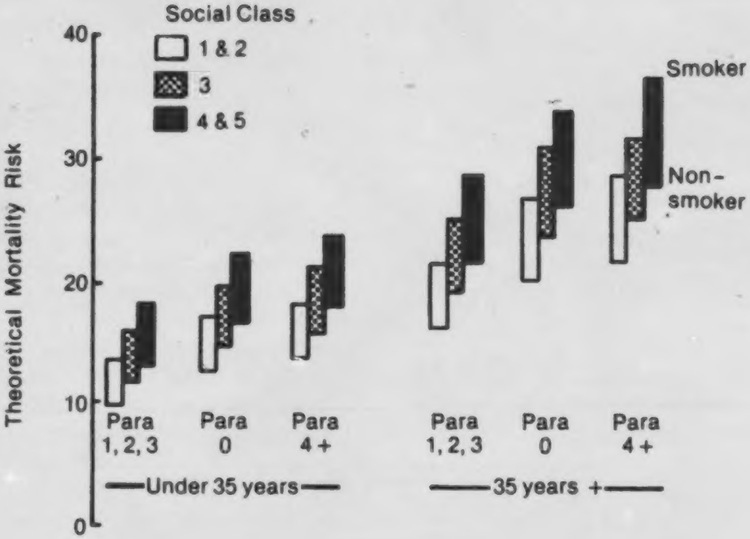

FIGURE 8.—Theoretical cumulative mortality risk according to smoking habit, in mothers of different age, parity, and social class groups

SOURCE: Butler, N.R. (15).

low-weight births, or anemia had an increased perinatal mortality risk of 70 to 100 percent when they were heavy smokers (88).

To help visualize the interacting effects of maternal smoking and of other factors on perinatal mortality risk, Butler has calculated theoretical mortality risks based on data from the British Perinatal Mortality Study. In Figure 8, perinatal mortality risks by social class, maternal age, and parity are arranged in order of increasing magnitude. The differences between smokers' and nonsmokers' risks are represented by the height of the bars, which varies depending on other risk factors (15).

These studies show that the risk of spontaneous abortion, of fetal death, and of neonatal death increases directly with increasing levels of maternal smoking during pregnancy. Studies of smoking during pregnancy show a range of perinatal mortality risk ratios (smokers versus nonsmokers) from a low of 1.01 to a high of 2.42. Variability between risk ratios in different study populations may be due to lack of comparability between smokers and nonsmokers in other respects, or to interaction between smoking and other pregnancy risk factors. Studies failing to take account of other important variables may show unusually high or unusually low risk ratios.

CAUSE OF DEATH

The increased perinatal mortality associated with maternal smoking is concentrated within a few cause-specific categories. Excess stillbirths have been associated with antepartum hemorrhage or abruptio placentae and with "unknown cause" (3,46). Excess neonatal deaths were associated with immaturity, asphyxia, atelectasis (23), and with the respiratory distress syndrome (3).

Meyer and Tonascia (87) analyzed fetal and neonatal deaths to identify causes of death which showed an excess if the mother smoked. Fetal and neonatal deaths by coded cause and maternal smoking habit are shown in Table 8. For each cause the observed numbers for smokers were compared with the number expected at nonsmoker rates. The differences between observed and expected numbers indicate the number of deaths in each category attributable to maternal smoking.

Fetal deaths showed a major smoking-related excess in the category of "unknown" cause and some increase from "anoxia" and "maternal cause." By contrast, neonatal deaths related to smoking were in the category of "prematurity alone," or in the related category of "respiratory difficulty." The tentative conclusion to be drawn here is that fetuses and neonates whose deaths were related to maternal smoking had no recognizable pathology, but had died *in utero* from anoxia, maternal cause, or unknown cause, or had suffered the consequences of preterm delivery.

Complications of Pregnancy and Labor

Studies have consistently found a direct relationship between maternal smoking level and the incidence of placenta previa, abruptio placentae, bleeding during pregnancy,and premature rupture of membranes (3,24,46,61,86,87,94,95,130,144,145). The association is independent of socioeconomic and racial background (144), parity (3) and many other factors (86) (Figure 8).

These complications carry with them a high risk of fetal and neonata loss, and are frequently cited as the cause of death among the offspring of women who smoke. Kullander and Kallen found a significant increase in the frequency of abruptio placentae among smokers' children dying before the age of 1 week (61). In a prospective study of 9,169 pregnancies by Goujard and colleagues, a large proportion of the increase in stillbirths among smokers was caused by abrutio placentae (46).

TABLE 8.—Fetal and neonatal deaths by coded cause and maternal smoking habit (Canadian English-speaking mothers)

Coded cause	Observed		Expected smoker*	Observed Expected difference	p+ value
	Nonsmoker	Smoker			
Fetal deaths					
Unknown	75	125	81.4	43.6	0.003
Malformations	32	24	34.7	−10.7	N.S.
Hemolytic disease	11	15	11.9	3.1	N.S.
Anoxia	16	29	17.4	11.6	N.S.
Maternal cause	31	45	33.u	11.3	N.S.
All others	8	13	8.7	4.3	N.S.
Total	173	251	187.9	63.1	0.003
Neonatal deaths					
Unknown	52	51	56.5	−5.5	N.S.
Malformations	22	24	23.9	0.1	N.S.
Hemolytic disease	7	8	7.6	0.4	N.S.
Respiratory difficulty	46	63	50.0	13.0	N.S.
Prematurity alone	33	65	35.8	29.2	0.005
Maternal cause	2	6	2.2	3.8	N.S.
All others	16	16	17.4	−1.4	N.S.
Total	178	233	193.3	39.6	0.06
Total Births	15,240	16,549			

N.S. = not significant.
*Based on nonsmoker rate.
p+ value derived from chi square based on a null hypothesis of no difference between smokers and nonsmokers.
SOURCE: Meyer, M.B. (87).

Naeye reviewed the clinical and postmortem material from the 3,897 fetal and infant deaths in the Collaborative Perinatal Project of the NINCDS (102) and reported an association between perinatal mortality rates caused by abruptio placentae and number of cigarettes smoked by the mother (95). Abruptio placentae was the underlying cause identified in 11 percent of all the deaths in this large study (94).

Analysis of data from the Ontario Perinatal Mortality Study corroborated these findings. Increasing levels of smoking resulted in a highly significant increase in the risks of placental abruptions, placenta previa, bleeding in pregnancy, and premature and prolonged rupture of membranes. Fetal and neonatal deaths were analyzed for associations between them and smoking-related excesses of various coded complications of pregnancy and labor. Although most diagnoses showed no association with excess mortality for smokers' babies, a few stood

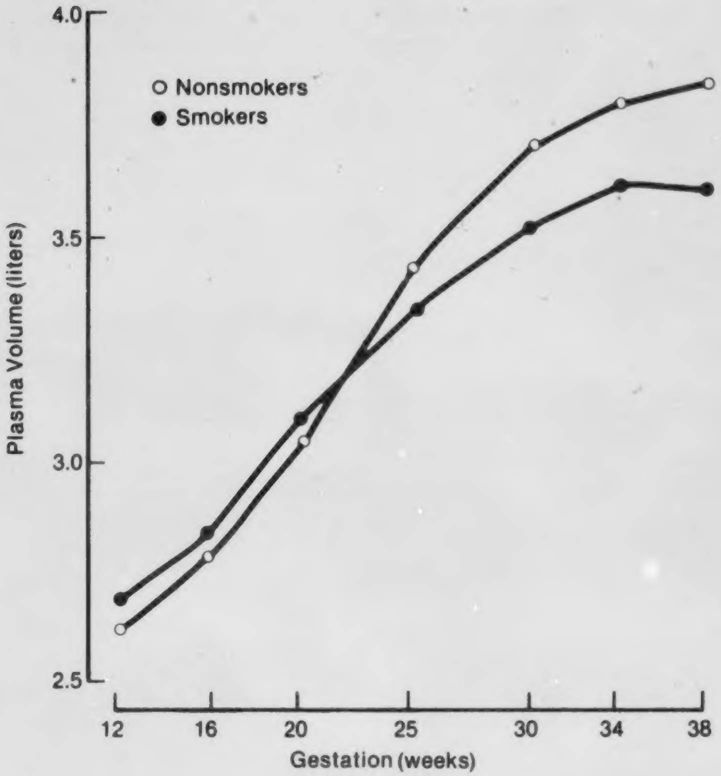

FIGURE 9.—Mean plasma volume in nonsmokers and smokers

SOURCE: Pirani, B.B.K. (117).

out as highly significant. Excess fetal deaths of smokers' babies were strongly associated with bleeding during pregnancy, either before (P = 0.01) or after (p = 0.0005) 20 weeks gestation. In other coded categories, a significant excess of fetal deaths occurred among smoking mothers with abruptio placentae (p=0.0001) or other obstetrical problems. Similar comparisons were made for neonatal deaths. A strong, significant relationship between smoking-related excess neonatal deaths and a history of bleeding before 20 weeks of gestation was found (p=0.0001). Other categories that showed significant increases of smoking-associated neonatal deaths were the admission status of rupture of membranes only, other obstetrical complications, and duration of rupture of membranes over 48 hours (87).

PREECLAMPSIA

Several published studies have reported that the incidence of preeclampsia is declining as the number of cigarettes smoked increases (109,145). Data from the British Prenatal Mortality Study were cross-tabulated by parity, severity of preeclampsia, and maternal smoking status. Smokers had lower rates of all grades of preeclampsia than nonsmokers, whether they were primiparae or multiparae (15). Andrews and McGarry showed that the inverse relationship between cigarette smoking and preeclamptic toxemia was independent of social class, maternal weight before pregnancy, and maternal weight gain during pregnancy (3). Despite this effect of smoking on the incidence of preeclampsia, there is a greatly increased risk of perinatal mortality if preeclampsia does develop in a smoker (3,34,129). Several authors have suggested that this negative association may be due to the hypotensive effect of thiocyanate, which is derived from the cyanide present in cigarette smoke and is regularly found in the blood of smokers (3,109). Because preeclampsia is predominantly a complication of first pregnancies, it is possible that the occasional finding of reduced rates of perinatal mortality in young, primiparous, light smokers who are otherwise healthy is due to this relationship.

Pirani and MacGillivray performed seven serial measurements from the end of the second trimester until term in 31 nonsmokers and 29 smokers. After 25 weeks gestation the plasma volume of smokers failed to keep pace with that for nonsmokers, the increases in volume being 25 percent less in smokers (Figure 9). Plasma volume and total body water expansion are related to birthweight, at least in primigravidas. After 30 weeks of gestation, total body water in smokers plateaued in contrast to nonsmokers, so that by term their body water volume increase was about 25 percent less. Serum heat-stable alkaline phosphatase levels in smokers significantly exceeded the concentration in nonsmokers from the 37th week of pregnancy onward. This enzyme is of placental origin, and cigarette smoking may contribute to this change by its effects on the placenta (117).

Whether the reduction in the incidence of preeclampsia with maternal smoking is due to the hypotensive effects of thiocyanate, to the reduced size of the baby, to a smaller increase in maternal blood volume, or to another process requires further study.

PRETERM DELIVERY, PREGNANCY COMPLICATIONS, AND PERINATAL MORTALITY BY GESTATION

Studies of large numbers of births to measure mean gestation by smoking habit have demonstrated differences of only a day or two. This finding led to the conclusion that maternal smoking does not affect gestation (14,52,74,102,146,159). On the other hand, abundant evidence has been presented that a smoking-related increase in preterm delivery plays an important role in the increased risk of neonatal death for infants of smokers.

When the proportion of preterm births is measured, rather than the mean gestation, smokers have shown consistently higher rates than nonsmokers, as illustrated in Table 9. In four studies in which all births and perinatal deaths were included, the risk of early delivery increased from 36 to 47 percent if the mother smoked, and 11 to 14 percent of all preterm births could be attributed to maternal smoking (3,15,38).

Figure 10, using data from the Ontario Perinatal Mortality Study, shows percentage distributions by gestational age of births to nonsmokers, light smokers, and heavy smokers, plotted on a semilogarithmic scale to emphasize differences between smoking-level groups in very preterm births. There is little difference between the means of these curves because the great majority of births occur around term in all groups. There is, however, a significant and dose-related increase in the proportions of preterm babies born to women who smoke. These preterm deliveries account for a small proportion of total births but for a large proportion of the deaths (82,146).

As previously reviewed, Meyer and Tonascia have related the excess fetal and neonatal mortality of smokers' infants and the excess incidence of pregnancy complications among women who smoke to the gestational age of occurrence, using a life-table approach. A starting population of all pregnancies *in utero* at 20 weeks was used to calculate the probabilities of fetal death, live delivery followed by survival or death, or the occurrence of a complication followed by fetal death or delivery. At 28 weeks (the next point defined by the data), the population at risk included those remaining *in utero* at that point. Figure 11 shows the probability of perinatal death during each period of gestational age starting at 20 weeks. Risks for smokers' infants were significantly greater in the earlier weeks, but not different after 38 weeks gestation (87,146).

A similar approach was applied to determine the risk by gestation of abruptio placentae, placenta previa, and premature rupture of membranes for smokers and nonsmokers. The risk of all these complications was higher for smokers throughout gestation, but in all the differences were most significant in the weeks of pregnancy from 20 to 32 or 36 weeks (87,146). The lower limit of 20 weeks was built into the study design, which included all single births of at least 20 weeks gestation (106,107).

These studies show that excess deaths of smokers' infants are

TABLE 9.—Preterm births by maternal smoking habit: relative and attributable risks, derived from published studies

Study	Smokers (proportion)	Preterm Births* per 100 Total Births Nonsmokers	Smokers	Relative Risk Smokers/Non-smokers	Attributable Risk %
Cardiff	.465	6.7	9.2	1.36	14
Great Britain	.274	4.7	6.9	1.47	11
Montreal	.432	7.7	10.6	1.38	14
Ontario	.435	7.4	10.1	1.36	14

*Cardiff and Ontario data are for <38 weeks. All others are for <37 weeks.

SOURCE: Andrews, J. (3), Campbell, J.M. (15), Fabia, J. (38), Meyer, M.B. (86), U.S. Department of Health, Education, and Welfare (146).

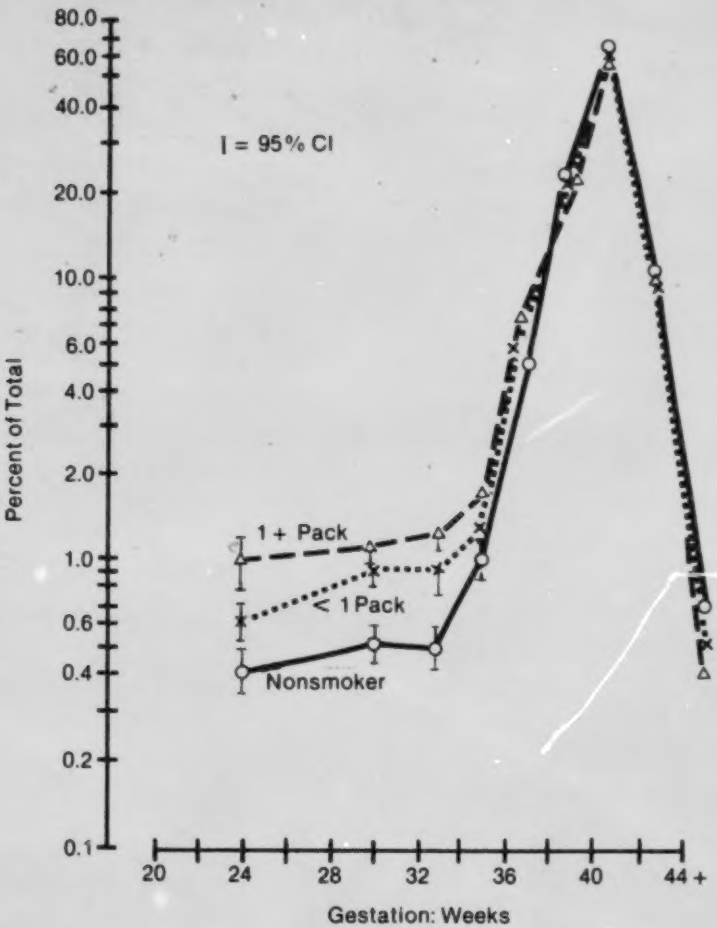

FIGURE 10.—Percentage distribution by weeks of gestation of births to nonsmokers, smokers of less than one pack per day, and smokers of one pack per day or more

SOURCE: Meyer, M.B. (82).

found mainly in the coded cause categories of "unknown" and "anoxia" for fetal deaths, and in the categories of "prematurity alone" and "respiratory difficulty" for neonatal deaths. This finding indicates that the excess deaths result not from abnormalities of the fetus or neonate, but from problems related to the pregnancy. Increasing levels of maternal smoking result in a highly significant increase in the risks of placental abruptions, placenta previa, bleeding early or late in pregnancy, premature and prolonged rupture of membranes, and preterm delivery, all of which carry high risks of perinatal loss. Although there is little effect of maternal smoking on mean gestation, the proportion of fetal deaths and live births that occur before term increases directly with maternal smoking level. Up to 14 percent of all preterm deliveries in the United States may be attributable to maternal smoking. According to the results of one large study, the most significant difference between smokers' and nonsmokers' risk of perinatal mortality and pregnancy complication occurs at the gestational ages from 20 to 32 or 36 weeks.

These findings lead to the conclusion that maternal smoking can be a direct cause of fetal or neonatal death in an otherwise normal infant. The immediate cause of most smoking-related fetal deaths is probably anoxia, which can be attributed to placental complications with antepartum bleeding in 30 percent or more of the cases. In other cases, the oxygen supply may simply fail from reduced carrying capacity and reduced unloading pressures for oxygen caused by the presence of carbon monoxide in maternal and fetal blood. Neonatal deaths occur as a result of the increased risk of early delivery among smokers, which may be secondarily related to bleeding early in pregnancy and premature rupture of membranes (146).

Long-Term Morbidity and Mortality

Studies of infant and child morbidity and mortality by the mother's smoking habits usually cannot distinguish between the effects of smoking during pregnancy and the effect of the infant's or child's passive exposure to cigarette smoke after birth. Several studies have found that hospitalization rates for pneumonia and bronchitis were higher during the first year of life for infants of smoking mothers (20,21,53). Rates in children were higher if the smoking parents also had cough and phlegm. Harlap and Davies found that the risk of contracting pneumonia or bronchitis in the first year of life more than doubled if the parents smoked more than 24 cigarettes a day (53).

A unique and important study of morbidity and mortality in smokers' and nonsmokers' children up to the age of five has now been published by Rantakallio (119). The experience up to age 5 of over 12,000 children born in 1966 in Northern Finland, comprising 96 percent of all births in two provinces, was ascertained through hospital and death records and questionnaires. Smok-

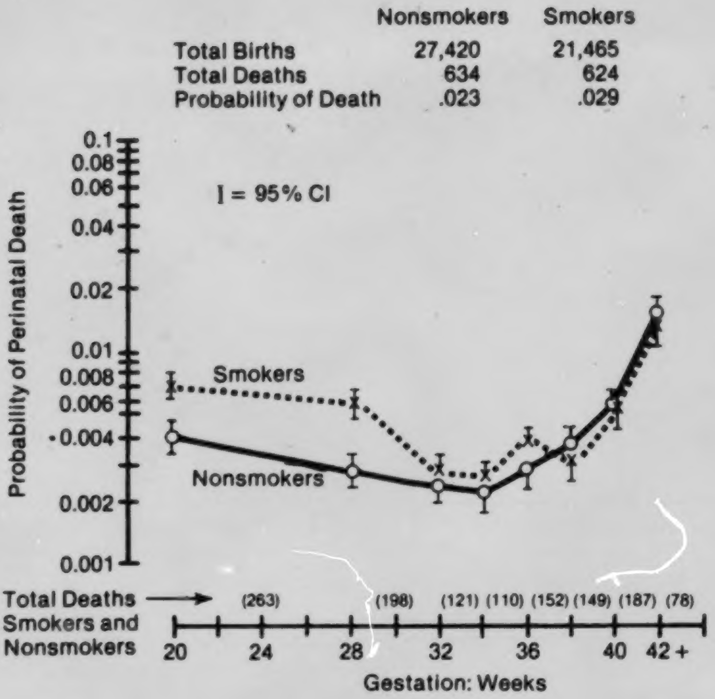

FIGURE 11.—**Probability of perinatal death for smoking and nonsmoking mothers, by period of gestational age (bars show 95% confidence intervals)**

SOURCE: Meyer, M.B. (27).

ing was rare in this population, and the smokers tended to be young and otherwise healthy. Fourteen percent of pregnant women smoked fewer than 10 cigarettes per day (mean number after the second month of pregnancy 3.9) and 3 percent smoked more than 10 cigarettes per day (mean number 12.2); the remaining 83 percent of the population were nonsmokers. It was therefore possible to remove the usual problems of confounding variables by close individual matching of 1,750 smokers to nonsmoking "controls". Matching factors included marital status, maternal age within 2 years, and place of residence, with the latter category including many socioeconomic variables to equalize the probable use of medical facilities and other differences. Although the author states that perinatal mortality did not show a statistically significant increase for smokers, rates were 24 per thousand for controls, 26 per thousand for light smokers, and 33 per thousand for "heavy" smokers (defined as smoking 10 plus cigarettes per day). These rates are similar to those found in other studies in which differences were statistically significant. Postneonatal mortality, from 28 days to 5 years, was higher for smokers' children with rates of 11.1 and 3.9 per thousand for smokers' and nonsmokers' children respectively. Overall death rates of 24.7 per thousand births in smoking women and 16.5 per thousand births in nonsmoking women were reported for children under the age of 5, of which 12.6 and 8.8 were neonatal.

In addition, the children of the smokers were hospitalized more frequently, had more visits to doctors, and had longer average durations of hospital stays than children of nonsmokers. Respiratory diseases caused significantly more hospitalizations among smokers' children. It is of great interest that the children born to a subgroup of women who stopped smoking during the last 3 months of pregnancy showed no increase of postneonatal mortality or morbidity up to the age of 5, compared with controls. However, these women had been very light smokers before quitting. Table 10, derived from Rantakallio's study, shows that the various outcomes measured show increasing rates of morbidity and mortality with increasing levels of smoking. However, it may not be possible to distinguish between the adverse effects of maternal smoking during pregnancy and the adverse effects on infants and children exposed to cigarette smoke in the home, because women who smoked during pregnancy probably also continued to smoke after pregnancy.

Because of the known carcinogenic potential of tobacco smoke and the evidence that benzo(a)pyrene reaches the placenta, Neutel and Buck investigated the relationship of maternal smoking during pregnancy to the incidence of cancer in children aged 7 to 10. A combined population of 89,302 births from the Ontario Perinatal Mortality Study and the British Perinatal Mortality Survey was used as a base population for a prospective study in which 65 cancer deaths and 32 cancer survivors were identified. For cancer of all sites, the children of smokers had a relative risk of 1.3, with 95 percent confidence limits of 0.8 to 2.2. A dose–response relationship was not observed. The numbers were not large enough to determine significant differences by site. Excess cancer rates for children of mothers who smoke and a possible dose-related progression were concentrated at ages 0 to 24 months, but these rates were based on small numbers of cases. The authors conclude that "although a significant excess is not demonstrable, a doubling of the cancer risk for children of smokers cannot be ruled out." Their equivocal re-

TABLE 10.—Long term effects of morbidity and mortality by level of maternal smoking

A. Mortality

	Nonsmokers		Light Smokers	Heavy Smokers
	Control 1	Control 2	(1-10 per day)	(10 + per day)
Number of children	1300	258	1302	252
Doctor visits per child (mean number)	.71	.61	.76	.83
Hospitalizations per child (mean number)	.19	.15	.22	.39
< Age 1	.14	.08	.17	.30
Age 1–5	.15	.17	.22	.25

B. Perinatal and postneonatal mortality (28 days to 5 years) per 100 births, by maternal smoking

	Nonsmokers	Smokers		
	Control	Light	Total	Heavy
Total births number	1844		1844	
Perinatal mortality per 1,000 births	23.9	25.7	26.0	32.6
Postneonatal mortality	3.9		11.1	
All mortality per 1,000 live births	16.5		24.7	

SOURCE: Rantakallio, P. (119).

sults were reported to encourage other workers to add to the data (99). This should certainly be done, with particular emphasis on the first 2 years of life.

Rantakallio, et al. also analyzed the use rates of ophthalmological services in their follow-up study of approximately 12,000 children, relating these rates of prenatal factors ascertained during pregnancy. The incidence of squint among smokers' children was 22.5 per thousand, compared with 11.5 per thousand among the children of matched, nonsmoking controls ($p < .05$). On the other hand, rates of dacryostenosis and of other congenital ocular malformations were higher among the children of controls. The authors state that squint was inversely correlated with birth weight and was more common among children with other diseases, especially nervous or mental diseases (121).

SUDDEN INFANT DEATH SYNDROME

Maternal smoking habits have been ascertained in several studies of the sudden infant death syndrome (SIDS). In all of these, an association has been found between maternal smoking during pregnancy and the incidence of sudden infant death. Steele and Langworth, in a study of 80 cases, each with two matched controls, which were traced back to the Ontario Perinatal Mortality Study population of 1960–61, found that sudden infant deaths were strongly associated with the frequency and level of maternal smoking during pregnancy ($p < .001$). Thirty-nine percent of the cases were nonsmokers versus 60 percent of controls; 36 percent of the cases and 27 percent of the controls smoked less than a pack per day; 24 percent of the cases and 10 percent of the controls smoked a pack per day or more. The habits of the remaining 1 to 2 percent of mothers were unknown (139).

Bergman and Wiesner studied 56 families who lost babies to the sudden infant death syndrome and 86 control families. They reported that a higher proportion of SIDS mothers smoked during pregnancy than controls (61 percent versus 42 percent), more smoked after pregnancy (59 percent versus 42 percent), and SIDS mothers smoked a significantly greater number of cigarettes than controls. These authors indicate that exposure to cigarette smoke (passive smoking) appears to enhance the risk for SIDS for reasons not yet known (8). However, whether prenatal or postnatal exposure is more important cannot be determined.

Naeye, et al., in their analysis of 125 SIDS victims from the population of the Collaborative Perinatal Project of the

NINCDS, stated: "The gestations that produced the SIDS victims were characterized by a greater frequency of mothers who smoked cigarettes and had anemia" than was true for the whole population of 53,721 infants or for a set of 375 controls matched for important factors (96). Rhead, commenting on studies published to date which demonstrate an increased incidence of maternal cigarette smoking in SIDS, states: "It is now . . . clear that maternal cigarette smoking contributes to an infant's risk of dying from SIDS" (123).

Analysis of data from the prospective study of 19,047 births to members of the Kaiser Foundation Health Plan (1960–1967) also showed a strong association of SIDS with maternal smoking. In the SIDS group, 70.6 percent of mothers smoked during pregnancy, compared with only 35.3 percent of mothers of babies who did not die of SIDS ($p < .001$). The relative risk of SIDS for smokers versus nonsmokers was 4.4 (67).

Mechanisms

Clues to the mechanisms by which smoking may increase the risk of pregnancy complications are available from pathological and physiological studies of placentas, membranes, blood vessels, circulatory patterns, and serum levels of substances important for cell and tissue integrity. For example, it is possible that placental changes in smokers that serve as adaptations to the hypoxic effects of carbon monoxide may also increase the risk of placental complications.

Christianson has reported findings from carefully standardized gross examinations of 7,651 placentas from smokers and nonsmokers. These examinations revealed that smokers' placentas were thinner and larger in their minimum diameter than those of nonsmokers. This significant change effectively increased the surface area of the smokers' placentas and must, therefore, have increased their area of attachment to the uterine wall. The distance from the edge of membrane rupture to the placental margin was also less for smokers, and significantly more smokers than nonsmokers had zero distance, which is consistent with the diagnosis of placenta previa (19). These findings suggest a possible mechanism to account for the significant dose-related increase in the frequency of the clinical diagnosis of placenta previa that accompanies maternal smoking (86). A similar increase in this condition occurs with increasing altitude (75).

Christianson's study also revealed that smokers had significantly more placental calcification, primarily of the maternal surface, and patchy subchorionic fibrin, as shown in Table 11.

TABLE 11.—Selected results of gross examinations of placentas from smokers and nonsmokers

	Percent of Placentas with Stated Condition					
	White			Black		
	Nonsmoker N=3,461	Smoker N=2,239	P	Nonsmoker N=1,300	Smoker N=652	P
Calcification	49.5	60.8	<.0001	43.5	59.0	<.0001
Patchy Subchorionic Fibrin	26.2	35.3	<.0001	30.8	37.0	<.01
Infarcts	24.6	22.3	<.05	14.4	14.5	NS
Thickness (mean cm)	2.16	2.12	<.001	2.11	2.06	<.01
Ratio of smallest diameter to thickness	8.19	8.40	<.001	8.39	8.68	<.01
Shortest distance, edge of rupture of membranes to placental margin (mean cm)	4.32	4.09	<.025	5.08	4.83	NS
Percent with zero distance	25.6	27.9	NS	18.6	20.3	<.05

SOURCE: Christianson, R.E. (19).

These changes are characteristic of maturation and aging of the placenta and occur as normal gestation proceeds; however, they occurred earlier in smokers than in nonsmokers (19). This finding is compatible with other manifestations of accelerated aging reported to be associated with cigarette smoking (28,108).

Asmussen compared placental vessels in smoking and nonsmoking mothers by electron microscopy. In the smoking group these vessels were characterized by subintimal edema with destruction of the intimal elastic membranes, a marked decrease in collagen content, and proliferation of myocytes. Asmussen postulated that similar damage may occur in the fetal and infant vascular system. To what extent such changes may predispose to the subsequent development of vascular disease remains unknown. The author regarded most of the changes observed in smokers' vessels as degenerative, but mentioned the possibility that the thickening of the basement membrane observed in smokers might be an attempt at repair (4,5). Naeye (93) has described an increased frequency of placental microscopic lesions associated with smoking. These include: cytotrophoblastic hyperplasia, obliterative endarteritis, stromal fibrosis, and small villous infarction. Smokers also demonstrated an increased frequency of necrosis and inflammation in the decidua capsularis and in the decidua basalis at the placental margin. Placental features observed less frequently in smokers' placentas were excessive syncytial knots and various thrombotic phenomena.

Naeye found increasing placental enlargement with smoking level, accompanied by decreasing birth weight and a consequent increase in the placental ratio. The author stated that "as smoking increased, placentas developed microscopic lesions characteristic of underperfusion of the uterus." Naeye's data showed positive trends with maternal smoking level for some findings and negative trends for others (93). Many of the changes cited were of low frequency in all groups, and no clear pattern of possible mechanisms of action emerged.

Other studies that may shed light on these complex interrelationships include the report by Goujard and colleagues that heavy alcohol consumption as well as smoking contributes to the risk of stillbirth caused by abruptio placentae. In a prospective survey of 9,169 women, the risk of stillbirth was 21 per 1,000 in smokers who were light or nondrinkers, 20 per 1,000 in nonsmoking drinkers of 45 ml equivalents or more of absolute alcohol per day, and 8.5 per thousand for nonsmokers who drank less than 45 ml per day. The small number of smokers who were also heavy drinkers had stillbirth rates of 50.5 per 1,000 (95 women with 5 stillbirths). The proportions of these deaths that

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were attributable to abruptio placentae increased with smoking and with drinking, based on data unadjusted for the effects of age, parity, and other factors (122).

More research is needed to define possible pathways of action by which the active components of cigarette smoke affect pregnancy complications that may lead, in turn, to fetal death or to preterm birth with or without survival.

Experimental Studies

TOBACCO SMOKE

Tobacco smoke contains more than 2,000 compounds including: carbon monoxide, oxides of nitrogen, ammonia, polycyclic aromatic hydrocarbons, hydrogen cyanide, vinyl chloride, and nicotine. For the pregnant woman and fetus the most important of these appear to be nicotine, carbon monoxide, and the polycyclic aromatic hydrocarbons.

NICOTINE

The effect of nicotine on sympathetic and parasympathetic ganglia, skeletal muscles, and the central nervous system is similar to that of acetylcholine. At all three sites it first stimulates, then depresses. Minute doses of nicotine stimulate the chemoreceptors of the carotid and aortic bodies, causing reflex hypertension. Nicotine also releases epinephrine from the adrenal medulla, thereby producing cardiovascular changes. Thus, it can produce widely differing effects depending upon the dosage and the particular site that is most sensitive to stimulation.

Nicotine rapidly crosses the placenta to affect the fetus (142). Relatively mature rhesus monkey fetuses respond to nicotine infusion with a rise in blood pressure, bradycardia, acidosis, hypercarbia, and hypoxia (141). Maternal nicotine administration in rats also has been shown to affect the fetal central nervous system and its response to electrical stimulation during the newborn period (56,78).

Quigley, et al. noted that in moderate to heavy smokers, after 34 weeks gestation, smoking two cigarettes in 10 minutes was associated with a 60 percent increase in maternal plasma norepinephrine and epinephrine and a 20 percent increase in serum cortisol concentrations (118). These changes also were associated with an increase in maternal pulse and blood pressure. Lehtovirta and Forss measured changes in placental intervillous blood flow using the 133 xenon method (66). Immediately after smoking, intervillous flow decreased 22 percent.

These data correlate with the studies of Resnik, et al. (122), showing nicotine-induced increases in catecholamines and decreased uterine blood flow in sheep, and of Haberman, demonstrating decreased uteroplacental blood flow in women, using thermography (48).

Sastry and his colleagues have carried out a series of studies on the effect of nicotine on the human placenta. Nicotine added to a calcium-containing medium caused a 33 percent increase in the rate of acetylcholine release from isolated placental villi (131). The authors postulated that this effect could account for the decrease in placental amino acid transport (125,154) produced by nicotine-mediated cholinergic blockade (105). Rowell and Sastry also demonstrated that nicotine caused a 41 percent decrease in uptake of alpha amino isobutyric acid in an experimental placental system (126). Their studies indicate that under normal circumstances acetylcholine exhibits a muscarinic effect facilitating placental amino acid uptake. Nicotine blockade of the facilitating effects of acetylcholine on amino acid uptake may result in fetal growth retardation (126). These data agree with the 1977 work of Crosby, et al. in humans (26).

Nicotine injection in rats results in prolonged gestation with lower than normal newborn weights. A possible cause of this prolonged gestation is nicotine-induced delay in ovum implantation. Yoshinaga, et al. tested this hypothesis, administering 7.5 mg nicotine tartrate twice daily from the morning of proestrus until the day of sacrifice on days 1 to 5 of pregnancy (161). The nicotine-injected animals demonstrated a delay of about 12 hours in ovum cleavage from the two- to the four-cell stage, and each step of development after the four-cell stage was thereby delayed. In addition, ovum entry into the uterus, blastocyst formation, shedding of the zona pellucida, and implantation were delayed. Nicotine injection also was associated with a "crowding" of implantation sites toward the tubal ends of the uterine horns.

During the preimplantation period the serum concentrations of progesterone, luteinizing hormone, and prolactin were lower, while the concentrations of estrogen and follicle stimulating hormone were higher than in control animals. These workers suggested that the delayed ovum implanation followed a delayed increase in progesterone secretion required to prepare the uterus for the implanting blastocyst, and that the delayed progesterone secretion results in part from nicotine-induced disturbed hypothalamus pituitary balance.

Hamosh, et al. observed that, while administration of 100 mg $kg^{-1}day^{-1}$ nicotine to pregnant rats from day 14 gestation onward failed to affect the mother or fetus, administration of 1 mg $kg^{-1}day^{-1}$

(a dose "comparable" to that of a 20 cigarette-per-day smoker) resulted in a decrease in litter size and an increase in stillbirth rate. Although administration of 100 mg $kg^{-1}day^{-1}$ nicotine failed to affect newborn birth weight by 12 days of age continued maternal nicotine administration resulted in a 9 percent decrease in body weight and a 40 percent decrease in weight of the stomach contents. These decreases presumably resulted from lower milk production by the nicotine-treated animals (51).

CARBON MONOXIDE (CO)

Carboxyhemoglobin concentrations of 4 to 5 percent are associated with numerous physiologic alterations in adults. Cigarette smoking raises the carboxyhemoglobin concentration 4 to 5 percent per pack smoked per day. Although CO diffuses across the placenta relatively slowly [the half time equals 1.5 to 2 hr (72)], fetal carboxyhemoglobin concentrations reflect those of the mother, and under steady state conditions are 10 to 15 percent higher than maternal levels (71). Elevated carboxyhemoglobin concentrations in the fetus are associated with decreased fetal blood oxygen tensions. These decreased oxygen tensions are associated with a redistribution of fetal blood flow to the brain, heart, and adrenal glands (146).

Carboxyhemoglobin concentrations have been described under several conditions of pregnancy. Davies, et al. (31) compared carboxyhemoglobin concentrations and "available oxygen" (a function of O_2 content in ml dl $blood^{-1}$) in women who stopped smoking for 48 hours during the last trimester of pregnancy, with women who did not stop smoking, and with nonsmoking women. In those women who stopped smoking, carboxyhemoglobin concentrations decreased. "Available oxygen" increased about 8 percent due both to an increase in functioning hemoglobin and a shift in the oxyhemoglobin saturation curve; this increase in "available oxygen" should contribute to improved fetal oxygenation.

Exposure of rabbits (6) and rats (39) to CO during gestation resulted in decreased fetal weights and increased perinatal mortality. Such CO-exposed newborn animals showed less activity as well as decreased lung weights and decreased concentrations of brain protein, DNA, and the neurotransmitters norepinephrine and serotonin (45). Cellular hypoxia is the final common pathway mediating the adverse effect of CO on the developing fetus.

Recent experimental studies have explored various aspects of CO-induced biochemical changes in the fetus and the newborn. Newby, et al. demonstrated a persistent effect of CO exposure in 8- and 13-day-old rats following a single 5-hour exposure to 1,500

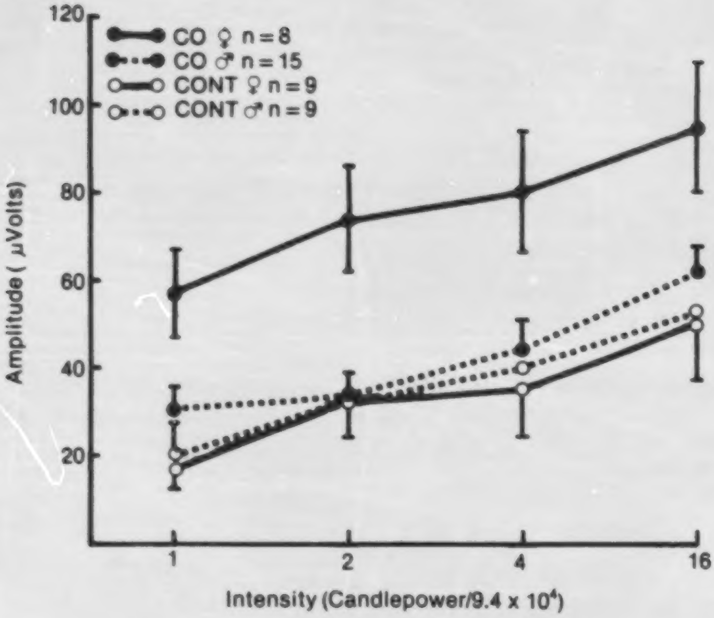

FIGURE 12.—Effect of prenatal CO upon peak-to-peak amplitudes of the first positiive to the first negative component of the flash evoked potential recorded from the rat visual cortex. Vertical bars represent ± standard error of the means

SOURCE: Dyer, R.S. (36).

parts per million (0.15 percent CO) (100). In these animals alpha methyl-p-tyrosine, a potent inhibitor of the enzyme tyrosine hydroxylase, was injected 1 hour before the CO exposure, and the extent of catecholamine depletion was taken as an index of the rate of catecholamine turnover. CO-treated rats showed increased steady state dopamine concentrations with decreased rates of dopamine turnover. In addition, the CO effect on dopamine turnover persisted for at least 3 to 6 weeks after a single exposure of 8-day-old rats. There was no CO effect on norepinephrine concentrations or turnover rates, and the effect was not produced in rats exposed to 8 percent oxygen instead of carbon monoxide. This is consistent with the data of Coyle and Campochiaro, which indicates that a maturational event occurs in the striatum of the 8-day-old rat (25). Whether this event represents the age of functional maturity, initiation of dopaminergic transmission, or maturation of cholinergic interneurons is unclear.

Prenatal CO exposure may have long-term consequences on central nervous system function. For instance, Dyer, et al. exposed female Long-Evans hooded rats to 150 ppm CO throughout pregnancy (36). At birth the litters and mothers were placed in room air without CO. On day 65 electrodes were placed in the young rats' skulls, and 2 weeks later visually evoked potentials were recorded. Figure 12 illustrates the effect of such prenatal exposure on the peak-to-peak amplitudes of the P1-N1 (first positive to first negative) component of the visual evoked potential from the cortex. Females showed a significant increase in P1-N1 amplitude at each of four flash intensities. Although the exact nature of this amplitude increase could not be determined, it suggests altered cell populations at the retinal, geniculate, and cortical levels, and may represent impaired inhibitory mechanisms, rendering other neurons more excitable.

The question of the posible teratogenicity of CO has never been resolved. Schwetz, et al. exposed mice to 250 ppm CO for 7 or 24 hours per day, from days 6 through 15 of gestation, and rabbits to the same concentration from days 6 through 18 (137). Blood carboxyhemoglobin concentration ranged from 10 to 15 percent. The fetuses of mice exposed to CO for 7 and 24 hours per day were slightly heavier and lighter, respectively, than those of the control animals. The only increase in teratogenic effects were minor skeletal variants such as extra lumbar ribs and spurs.

POLYCYCLIC AROMATIC HYDROCARBONS

The polycyclic aromatic hydrocarbons (PAH), such as benzo(a)pyrene, are widely distributed mutagens and carcinogens. These substances, produced by incomplete combustion of organic material, are important constituents of tobacco smoke. Exposure of cells to PAH induces the enzyme, aryl hydrocarbon hydroxylase. The inducibility of this enzyme system has been used by some workers to demonstrate, indirectly, that benzo(a)pyrene and other polycyclic hydrocarbons reach the placenta and fetus.

The placental concentration of benzo(a)pyrene is highly correlated with the amount which a pregnant woman smokes (97, 111). In pregnant rats exposed to this substance higher doses were required to induce enzyme activity in the fetus as compared with the dose required to stimulate placental enzyme activity (153), suggesting that the placenta may protect the fetus from these substances. However, the placenta is not impermeable to benzo(a)pyrene (134). The placenta is involved in complex hormonal interrelations between mother and fetus, and oxidative enzyme pathways in the placenta are important in maintaining hormonal and nutrient balance for normal fetal development. The hydroxylation of polycyclic hydrocarbons and the active transport of various compounds by trophoblast cells may share common enzyme systems. Thus, the induction of various enzymes by polycyclic hydrocarbons may interfere with normal transport systems.

Another unanswered question concerns the carcinogenic risk for progeny exposed *in utero* to polycyclic aromatic hydrocarbons. The offspring of mice that were injected with benzo(a)pyrene late in gestation showed an increased incidence of neoplasms of the lungs, liver, and mammary glands (101). Pelkonen, et al. determined that placental aryl hydrocarbon hydroxylase activity correlated closely with both the amount the mother smoked and newborn weight (112). These authors suggested that the placental concentration of this enzyme may be used as a measure of fetal exposure to maternal cigarette smoking. Vaught, et al. also reported much higher aryl hydrocarbon hydroxylase activity in the placental microsomes of smokers compared with nonsmokers (148).

Although currently available data do not allow a quantitative assessment of the genetic risk to man from cigarette smoking, such risk may occur since so many components of cigarette smoke are mutagens (as well as carcinogens) (11). Male cigarette smokers may have an increased number of abnormal spermatozoa (150). Paternal and maternal chromosomal aberrations (103) and sister chromatid exchanges may be increased in smokers (62). Because the proportion of smokers in the population is so high (between 30 and 50 percent), even a relatively weak mutagenic effect could have a significant effect on the gene pool (11).

OTHER COMPONENTS

Cyanide, another constituent of cigarette smoke, may contribute to retarded infant growth and increased perinatal mortality. Smokers have increased levels of cyanide and thiocyanate in body fluids. Serum concentrations of vitamin B_{12}, used in cyanide metabolism, are decreased as well. Several workers have recorded increased thiocyanate concentrations in both women who smoke and in their fetuses (2,140,154). Pettigrew, et al. compared cyanide and thiocyanate concentrations in smokers and nonsmokers, matched for age, height, parity, and socioeconomic status (116). Cyanide and thiocyanate concentrations were two to four times greater in the blood and urine of smokers and in the urine of smokers' infants as compared with controls. Meberg, et al. reported that thiocyanate concentrations were correlated with cigarette consumption and inversely correlated with birth weight (81).

Cadmium, another constituent of tobacco smoke, is concentrated in the placenta of smokers (124). Webster exposed pregnant mice to 10 to 40 ppm cadmium and noted an inverse correlation between cadmium concentration and fetal weight (152).

Lauwerys, et al. examined the effects of epidemiology factors on heavy metal and CO concentrations in the blood, placenta, and fetus of smoking women (65). Cadmium concentrations in maternal blood were twofold greater than concentrations in fetal blood, suggesting that the placenta acts as a barrier to this metal. They reported a correlation between maternal cadmium and carboxyhemoglobin concentrations (13,65). They also found that the cadmium concentration of smokers' placentas was about 25 percent greater than in a control group and that the placental cadmium concentration exceeded that of maternal blood about tenfold (124).

Fertility

Fertility results from the successful completion of a complex step-wise process beginning with gametogenesis (sperm and egg production), continuing through gamete release (ejaculation and ovaluation), gamete interaction (fertilization), conceptus transport through the fallopian tube into the uterus, and ending with implantation of the embyro into the endometrial wall. An adverse effect of smoking on any of these steps may impair fertility.

SMOKING AND REPRODUCTION IN WOMEN

Several epidemiologic studies have suggested that smoking decreases fertility in women (50,115,143,149). The retrospective study of Tokuhata demonstrated that 21 percent of women who regularly smoked cigarettes were infertile while only 14 percent of those who never used tobacco regularly were infertile (143). After several characteristics (cause of death, age at and year of death, education, occupation and frequency of marriage as well as husbands' smoking habits, education and occupation) were controlled, a 46 percent excess of infertility was found in women who smoked.

In a study on the return of fertility after discontinuing contraception, Vessey, et al. found a suggested reduction in fertility among women smoking 15 or more cigarettes per day (149). Pettersson, et al. found a tendency toward a greater prevalence of secondary amenorrhea among smokers (4.8/100 women) than among nonsmokers (3.7/100 women) (115). Hammond found that 49 percent of the nonsmoking women between 40 and 49 years had regular menses while only 40 percent of those smoking more than one pack a day had a regular menses (50). Conversely only 18 percent of nonsmokers had irregular menses while 24 percent of those smoking one or more packs of cigarettes per day said they had irregular menses. Smoking women were also more likely to have an unusual vaginal discharge and vaginal bleeding than nonsmokers. Experimental studies have demonstrated alterations in luteinizing hormone release and a decreased ovulatory response in rats exposed to tobacco smoke (76).

The effect of smoking on ovulation may result from direct effects of nicotine on the hypothalamus or pituitary. This would alter the release of gonadotropin releasing hormones from the hypothalamus or impair the pituitary response to releasing hormones.

SMOKING AND AGE OF MENOPAUSE

Substantial data demonstrate that smoking lowers the age of spontaneous menopause (7,9,27,58,68,69). The recent study by Jick, et al. revealed a dose dependent decrease in the age of menopause in smoking women who live in Sweden and the United States (58). The median age of menopause in nonsmokers was 50; among those smoking one-half pack/day it was 49; in those smoking 1 or more pack/day, it was 48. Similar studies have been published indicating an earlier onset of menopause in smoking women in the United States (29), in England (7), in Germany (9), and in Sweden (68,69). The mechanism of early menopause in smokers may be related to ovotoxins in cigarette smoke (37) or to toxic alterations in the hormonal regulatory mechanisms controlling the hypothalamic–pituitary–ovarian axis (76). One group of ovotoxins may be polycyclic aromatic hydrocarbons which have been demonstrated to be metabolized by ovarian enzymes to toxic products which destroy oocytes in rat and mouse ovaries (47,79).

Evidence collected by Daniell (29) and Lindquist (68) suggest that the earlier menopause of smokers is not related to weight differences between smokers and nonsmokers but is a direct result of some component of cigarette smoke.

SMOKING AND REPRODUCTION IN MEN

Spermatogenesis, sperm morphology, sperm motility (17,64,133,150) and androgen secretion (12,113) appear to be altered in men who smoke. Viczian (150) has demonstrated decreased sperm density, a cigarette-dose-dependent decrease in sperm motility, and a cigarette-dose-dependent increased abnormal sperm morphology among smokers.

In metabolic studies of alcoholic men admitted to a clinical research center, an inverse relationship between number of cigarettes smoked and reduction of testosterone levels was seen (113). Briggs (12) has reported lower plasma testosterone among smoking men compared to matched nonsmoking controls and has shown that cessation of smoking resulted in increased testosterone levels in these men. Wintermitz and Quillen (158) in a study on the acute effects of smoking in men demonstrated increases in plasma cortisol and growth hormone during the smoking period. Growth hormone returned to the presmoking level shortly after the smoking period, and cortisol fell gradually to the presmoking level by 90 minutes after cessation of smoking. Urinary catecholamines were higher on the smoking day than the nonsmoking day. No acute changes were observed in gonadotropins or testosterone in these men. These studies demonstrate stimulatory effects of smoking on growth hormone and cortisol.

Studies in experimental animals have also shown that tobacco smoke impairs spermatogenesis (37,151). Smoking also lowers sexual activity in male rats (18).

These data suggest two possible mechanisms of action of smoking on male reproduction. A component of cigarette smoke may have a direct action on the testes, disrupting gamete production. This would be consistent with the suggested effect of cigarette smoke on the ovary. In addition, cigarette smoke is known to contain compounds which are mutagenic (59). Alternatively, cigarette smoke may interfere with the regulatory mechanisms controlling the hypothalamic–pituitary–testicular axis.

FERTILIZATION AND CONCEPTUS TRANSPORT

The effect of smoking on sperm-egg interaction (fertilization) has not been studied in mammalian species. Evidence from sub-mammalian species demonstrates that nicotine promotes polyspermy (the entrance of more than one sperm into the oocyte) (73). Polyspermy would result in abnormal embryonic development and early abortion, which is one known effect of smoking (60).

The effect of smoking on conceptus transport in the fallopian tube or entry into the uterus is unknown; however, some evidence suggests that smoking can alter the amplitude and tone of contractions measured during the Rubin uterotubal insufflation test (a combined measure of uterotubal junction and tubal patency) (98), suggestive that smoking may alter conceptus transport in the fallopian tube or its entrance into the uterus.

In summary, cigarette smoking appears to exert an adverse effect on fertility. Further studies are needed to quantify the effects, identify etiologic agent(s), and define the mechanism(s) of action.

Summary

1. Babies born to women who smoke during pregnancy are, on the average, 200 grams lighter than babies born to comparable nonsmoking women.

2. The relationship between maternal smoking and reduced birth weight is independent of all other factors that influence birth weight including race, parity, maternal size, socioeconomic status, and sex of child; it is also independent of gestational age.

3. There is a dose-response relationship between maternal smoking and reduced birth weight; the more the woman smokes during pregnancy, the greater the reduction in birth weight.

4. If a woman gives up smoking early during pregnancy, her risk of delivering a low-birth-weight baby approaches that of a nonsmoker.

5. The ratio of placental weight to birth weight increases with increasing levels of maternal smoking, reflecting a considerable decrease in mean birth weight and a slight increase in mean placental mass; this may represent an adaptation to relative fetal hypoxia.

6. The pattern of fetal growth retardation that occurs with maternal smoking is a decrease in all dimensions including body length, chest circumference, and head circumference.

7. Maternal smoking during pregnancy may adversely affect the child's long-term growth, intellectual development, and behavioral characteristics.

8. Maternal smoking during pregnancy exerts a direct growth-retarding effect on the fetus; this effect does not appear to be mediated by reduced maternal appetite, eating or weight gain.

9. The risk of spontaneous abortion, fetal death, and neonatal death increases directly with increasing levels of maternal smoking during pregnancy; interaction of maternal smoking with other factors which increase perinatal mortality may result in an even greater risk.

10. Excess deaths of smokers' infants are found mainly in the coded cause categories of "unknown" and "anoxia" for fetal

deaths, and the categories of "prematurity alone" and "respiratory difficulty" for neonatal deaths; this suggests that the excess deaths are due to problems of the pregnancy, rather than to abnormalities of the fetus or neonate.

11. Increasing levels of maternal smoking result in a highly significant increase in the risk of abruptio placentae, placenta previa, bleeding early or late in pregnancy, premature and prolonged rupture of membranes, and preterm delivery—all of which carry high risks of perinatal loss.

12. Although there is little effect of maternal smoking on mean gestation, the proportion of fetal deaths and live births that occur before term increases directly with maternal smoking level. Up to 14 percent of all preterm deliveries in the United States may be attributable to maternal smoking.

13. The incidence of preeclampsia is decreased among women who smoke during pregnancy; however, if preeclampsia develops in a smoking woman, the risk of perinatal mortality is markedly increased compared to preeclamptic nonsmokers.

14. An infant's risk of developing the "sudden infant death syndrome" is increased by maternal smoking during pregnancy.

15. There are insufficient data to support a judgement on whether maternal and/or paternal cigarette smoking increases the risk of congenital malformations.

16. Infants and children born to smoking mothers may experience more long-term morbidity than those born to nonsmoking mothers; however, studies usually cannot distinguish between the effects of smoking during pregnancy and the effects of the infant's or child's passive exposure to cigarette smoke after birth.

17. Studies in women and men suggest that cigarette smoking may impair fertility.

18. Experimental studies on tobacco smoke, nicotine, carbon monoxide, polynuclear aromatic hydrocarbons, and other constituents of smoke help define pathways by which maternal smoking during pregnancy may exert its aforementioned effects.

References

(1) ALBERMAN, E., CREASY, M., ELLIOTT, M., SPICER, C. Maternal factors associated with fetal chromosal anomalies in spontaneous abortions. British Journal of Obstetrics and Gynecology 83: 621-627, August 1976.

(2) ANDREWS, J. Thiocyanate and smoking in pregnancy. British Journal of Obstetrics and Gynecology 80: 810-814, 1973.

(3) ANDREWS, J., MCGARRY, J. M. A community study of smoking in pregnancy. Journal of Obstetrics and Gynecology of the British Commonwealth 79(12): 1057-1073, December 1972.

(4) ASMUSSEN, I. Arterial changes in infants of smoking mothers. Postgraduate Medical Journal 54: 200-204, March 1978.

(5) ASMUSSEN, I. Ultrastructure of human umbilical veins. Acta Obstetrica et Gynecologica Scandinavica, Supplement 57(3): 253-255, 1978.

(6) ASTRUP, P., OLSEN, H. M., TROLLE, D., KJELDSEN, K. Effect of moderate carbon-monoxide exposure on fetal development. Lancet 2: 1220-1222, December 9, 1972.

(7) BAILEY, A., ROBINSON D., VESSEY, M. Smoking and age of natural menopause. Lancet 2: 722, 1977.

(8) BERGMAN, A. B., WIESNER, L. A. Relationship of passive cigarette-smoking to sudden infant death syndrome. Pediatrics 58(5): 665-668, November 1976.

(9) BERNHARD, P. Die Wirkung des Rauchers auf Fran und Mutter. (The effect of smoking on women and mothers.) Munchner Medizinische Wochenschrift 104: 1826-1831, 1962.

(10) BORLEE, I., LECHAT, M. F. Resultats d'une enquête sur les malformations congenitales dans le Hainaut. (Results of a study on congenital malformations in Hainaut.) Belges de Medicine Sociale, Hygiene, Medecine du Travail et Medecine Legale (Brussels) 36(2): 77-99, February 1979.

(11) BRIDGES, B. A., CHELMMESEN, J., SUGIMURA, T. Cigarette smoking—does it carry a genetic risk? Mutation Research 65: 71-81, 1979.

(12) BRIGGS, W. H. Cigarette smoking and infertility in men. Medical Journal of Australia 1(12): 616-617, 1973.

(13) BRITISH MEDICAL JOURNAL. Cigarette smoking and spontaneous abortion. British Medical Journal 1 (6108): 259-260, February 4, 1978.

(14) BUNCHER, C. R. Cigarette smoking and duration of pregnancy. American Journal of Obstetrics and Gynecology 103(7): 942-946, April 1, 1969.

(15) BUTLER, N. R., ALBERMAN, E. D. (Editors). Perinatal Problems. The Second Report of the 1958 British Perinatal Mortality Survey. London, E. and S. Livingston, Ltd., 1969, pp. 36-84.

(16) BUTLER, N. R., GOLDSTEIN, H. Smoking in pregnancy and subsequent child development. British Medical Journal 4: 573-575, December 8, 1973.

(17) CAMPBELL, J. M., HARRISON, K. L. Smoking and infertility. Medical Journal of Australia 1(8): 342-343, 1979.

(18) CENDRON, H., VALLERY-MASSON, J. Tabac et Comportement Sexuel Chez L'Homme. (Tobacco and Sexual Behavior of Men.) Vie Medicale 52(25): 3027-3030, July 1971.

(19) CHRISTIANSON, R. E. Gross differences observed in the placentas of smokers and nonsmokers. American Journal of Epidemiology 110(2): 178-187, August 1979.

(20) COLLEY, J. R. T., Respiratory symptoms in childhood and parental smoking and phlegm production. British Medical Journal 2: 201-204, 1974.

(21) COLLEY, J. R. T., HOLLAND, W. W., CORKHILL, R. T. Influence of passive smoking and parental phlegm on pneumonia and bronchitis of early childhood. Lancet 2: 1031-1034, 1978.

(22) COMSTOCK, G. W., LUNDIN, F. E., JR. Parental smoking and perinatal mortality. American Journal of Obstetrics and Gynecology 98(5): 708-718, July 1, 1967.

(23) COMSTOCK, G. W., SHAH, F. K., MEYER, M. B., ABBEY, H. Low birth weight and neonatal mortality rate related to maternal smoking on

socioeconomic status. American Journal of Obstetrics and Gynecology 111(1): 53-59, September 1, 1971.

(24) COPE, I., LANCASTER, P., STEVENS, L. Smoking in pregnancy. Medical Journal of Australia 1: 673-677, April 7, 1973.

(25) COYLE, J. T., CAMPOCHIARO, P. Ontogenesis of dopaminergic-cholinergic interactions in the rat striatum: a neurochemical study. Journal of Neurochemistry 27: 673-678, 1976.

(26) CROSBY, W. M., METCOFF, J., COSTILOE, J. P., MAMEESH, M. SANDSTEAD, H. H., JACOB, R. A., MCCLAIN, P. E., JACOBSON, G., REID, W., BURNS, G. Fetal malnutrition: an appraisal of correlated factors. American Journal of Obstetrics and Gynecology 128: 22-31, 1977.

(27) DANIELL, H. W. Osteoporosis of the slender smoker. Archives of Internal Medicine 136: 298-304, 1976.

(28) DANIELL, H. W. Smokers' wrinkles. Annals of Internal Medicine 75:873-880, 1971.

(29) DANIELL, H. W. Smoking, obesity, and the menopause. (Letter). Lancet 2(8085): 373, August 12, 1978.

(30) DAVIE, R., BUTLER, N., GOLDSTEIN, H. From Birth to Seven. The Second Report of the National Child Development Study (1958 Cohort). London, Longman, in association with the National Children's Bureau, 1972 198 pp.

(31) DAVIES, D. P., GRAY, O. P., ELLWOOD, P. C., ABERNETHY, M. Cigarette smoking in pregnancy: associations with maternal weight gain and fetal growth. Lancet 1: 385-387, February 21, 1976.

(32) DENSON, R., NANSON, J. L., MCWATTERS, M. A. Hyperkinesis and maternal smoking. Canadian Psychiatric Association Journal 20(3): 183-187, April 1975.

(33) DONOVAN, J. W. Effect on child of maternal smoking during pregnancy. (Letter). Lancet 1: 376, February 17, 1973.

(34) DUFFUS, G. M., MACGILLIVRAY, I. The incidence of preeclamptic toxaemia in smokers and nonsmokers. Lancet 1(7550): 994-995, May 11, 1968.

(35) DUNN, H. G., MCBURNEY, A. K., INGRAM, S., HUNTER, C. M. Maternal cigarette smoking during pregnancy and the childs subsequent development: 1. Physical growth to the age of 6½ years. Canadian Journal of Public Health 67: 499-505, November/December 1976.

(36) DYER, R. S., ECCLES, C. U., SWARTZWELDER, H. S., FECHTER, L. D., ANNAU, Z. Prenatal carbon monoxide and adult evoked potentials in rats. Journal of Environment, Science and Health C13: 107-120, 1979.

(37) ESSENBERG, J. M., FAGAN, L., MALERSTEIN, A. J. Chronic poisoning of the ovaries and testes of albino rats and mice by nicotine and cigarette smoke. Western Journal of Surgery, Obstetrics and Gynecology 59: 27-32, 1951.

(38) FABIA, J. Cigarettes pendant la grossesse, poids de naissance et mortalite perinatale (Cigarette smoking during pregnancy, birth weight and perinatal mortality.) Canadian Medical Association Journal 109: 1104-1109, December 1, 1973.

(39) FECHTER, L. D., ANNAU, Z. Toxicity of mild prenatal carbon monoxide exposure. Science 197 (4304): 680-682, April 12, 1977.

(40) FEDRICK, J. Factors associated with low birth weight of infants delivered in term. British Journal of Obstetrics and Gynecology 85(1): 1-7, January 1978.

(41) FEDRICK, J., ALBERMAN, E. D., GOLDSTEIN, H. Possible

(60) KLINE, J., STEIN, Z. A., SUSSER, M., WARBURTON, D. Smoking: A risk factor for spontaneous abortion. New England Journal of Medicine 297(15): 793-796, October 13, 1977.

(61) KULLANDER, S., KAELLEN, B. A prospective of smoking and pregnancy. Acta Obstetrica et Gynecologica Scandinavica 50(1): 83-94, 1971.

(62) LAMBERT, B., LINDBLAD, A., NORDENSKYJOLD, M., WERELIUS, B. Increased frequency of sister chromatid exchanges in cigarette smokers. Hereditas 88: 147-149, 1978.

(63) LANCET. Smoking and intrauterine growth. Lancet 1(8115): 536-537, March 10, 1979.

(64) LASZLO, V. A dohanyzas karos hatasai a gestatios folyamatokra. (The deleterious effects of smoking on the sequences of gestation.) Magyan Noorvosok Lapja 32(2): 163-167, March 1969.

(65) LAUWERYS, R., BUCHET, J. P., ROELS, H., HUBERMONT, G. Placental transfer of lead, mercury, cadmium, and carbon monoxide in women: I. Comparison of the distributions of the biological indices in maternal and umbilical cord blood. Environmental Research 15: 278-289, 1978.

(66) LEHTOVIRTA, P., FORSS, M. The acute effect of smoking on intervillous blood flow of the placenta. British Journal of Obstetrics and Gynecology 85: 729-731, 1978.

(67) LEWAK, N. VAN DEN BERG, B., BECKWITH, J. B. Sudden infant death syndrome risk factors. Clinical Pediatrics 18(7): 404-411, July 1979.

(68) LINDQUIST, O., BENGTSSON, C. Menopausal age in relation to smoking. Acta Medica Scandinavica 205: 73-77, 1979.

(69) LINDQUIST, O., BENGTSSON, C. The effect of smoking on menopausal age. Maturitas 1: 171-173, 1979.

(70) LONGO, L. D. Carbon monoxide: Effects on oxygenation of the fetus in utero. Science 194: 523-525, October 29, 1976.

(71) LONGO, L. D. Carbon monoxide in the pregnant mother and fetus and its exchange across the placenta. Annals of the New York Academy of Sciences 174(Article 1): 313-341, October 5, 1970.

(72) LONGO, L. D. The biological effects of carbon monoxide on the pregnant woman, fetus, and newborn infant. American Journal of Obstetrics and Gynecology 129(1): 69-103, September 1, 1977.

(73) LONGO, F. J., ANDERSON, E. The effects of nicotine on fertilization in the sea urchin, Ardacia punctulata. Journal of Cell Biology 46(2): 308-325, August 1970.

(74) LOWE, C. R. Effect of mothers' smoking habits on birth weight of their children. British Medical Journal 2:673-676, October 10, 1959.

(75) MCCLUNG, J. Effects of High Altitude on Human Birth. Observations on Mothers, Placentas, and the Newborn in Two Peruvian Populations. Cambridge, Harvard University Press, 1969, 150 pp.

(76) MCLEAN, B. R., RUBEL, A., NIKITOVITCH-WINER, M. B. The differential effects of exposure to tobacco smoke on the secretion of luteinizing hormone and prolactin in the proestrous rat. Endocrinology 100: 1561-1570, 1977.

(77) MACMAHON, B., ALPERT, M. SALBER, E. J. Infant weight and parental smoking habits. American Journal of Epidemiology 82(3): 247-261, November 1966.

(78) MARTIN, J. C., BECKER, R. F. The effects of maternal nicotine absorption or hypoxic episodes upon appetitive behavior of rat offspring. Developmental Psychobiology 4(2): 133-147, 1971.

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teratogenic effect of cigarette smoking. Nature 231: 529-530, June 25, 1971.

(42) FERGUSSON, D. M. Smoking during pregnancy. New Zealand Medical Journal 89(628): 41-43, January 24, 1979.

(43) GARN, S.M., HOFF, K., McCABE, K.D. Is there nutritional mediation of the "smoking effect" on the fetus? American Journal of Clinical Nutrition 32: 1181-1187, June 1977.

(44) GARN, S.M., SHAW, H. A., McCABE, K.D. Effect of maternal smoking on weight and weight gain between pregnancies. American Journal of Clinical Nutrition 31(8): 1302-1303, August 1978.

(45) GARVEY, D. J., LONGO, L. D. Chronic low level maternal carbon monoxide exposure and fetal growth and development. Biology of Reproduction 19: 8-14, 1978.

(46) GOUJARD, J., RUMEAU, C., SCHWARTZ, D. Smoking during pregnancy, stillbirth and abruptio placentae. Biomedicine 23: 20-22, 1975.

(47) GUIYAS, B. G., MATTISON, D. R. Degeneration of mouse oocytes in response to polycyclic aromatic hydrocarbons. Anatomical Record 193: 863-882, 1975.

(48) HABERMAN-BRUESCHKE, J. D., BRUESCHKE, E. E., ISARD, H. J., GERSHON-COHEN, J. Effect of smoking on skin temperature as demonstrated by the thermograph. Journal of the Albert Einstein Medical Center 13(3): 205-210, July 1965.

(49) HAJERI, H., SPIRA, A., FRYDMAN, R., PAPIERNIK-BERKHUER, E. Smoking during pregnancy and maternal weight gain. Journal of Perinatal Medicine 7 (1): 33-38, 1979.

(50) HAMMOND, E. C. Smoking in relation to physical complaints. Archives of Environmental Health. 3: 28-46, 1961.

(51) HAMOSH, M., SIMON, M. R., HAMOSH, P. Effect of nicotine on the development of fetal and suckling rats. Biology of the Neonate 35: 290-297, 1979.

(52) HARDY, J. B., MELLITS, E. D. Does maternal smoking during pregnancy have a long-term effect on the child? Lancet 2: 1332-1336, December 23, 1972.

(53) HARLAP, S., DAVIES, A. M. Infant admissions to hospital and maternal smoking. Lancet 1: 527-532, 1974.

(54) HIMMELBERGER, D. U., BROWN, B. W., COHEN, E. N. Cigarette smoking during pregnancy and the occurrence of spontaneous abortion and congenital abnormality. American Journal of Epidemiology 108(6): 470-479, December 1978.

(55) HOLLINGSWORTH, D. R., MOSER, R. J., CARLSON, J. W., THOMPSON, K. T. Abnormal adolescent primiparous pregnancy: Association of race, human chorionic somatomammotropin production, and smoking. American Journal of Obstetrics and Gynecology 126(2): 230-237, September 15, 1976.

(56) HUDSON, D. B., MEISAMI, E., TIMIRAS, P. S. Brain development in offspring of rats treated with nicotine during pregnancy. Experientia 29(3): 286-288, 1973.

(57) JARVINEN, P. A., OSTERLUND, K. Effect of smoking during pregnancy on the fetus, placenta and delivery. Annales Paediatriae Fenniae 9: 18-26, 1963.

(58) JICK, H., PORTER, J., MORRISON, A. S. Relation between smoking and age of natural menopause. Lancet 1: 1354-1355, 1977.

(59) KIER, L. D., YAMASAKI, E., AMES, B. Detection of mutagenic activity in cigarette smoke condensates. Proceedings of the National Academy of Sciences U.S.A. 71: 4159-4163, 1974.

(79) MATTISON, D. R., THORGEIRSSON, S. S. Ovarian aryl hydrocarbon hydroxylase activity and primordial oocyte toxicity of polycyclic aromatic hydrocarbons in mice. Cancer Research 39: 3471-3475, 1979.

(80) MAU, G., NETTER, P. Die Auswirkungen des viaterlichen zigarettenkongums aug die perintale sterblichkeit und lie missbildungshaufigkeit. (The effects of paternal cigarette smoking on perinatal mortality and on incidence of malformations.) Deutsche Mediziniche Wockenschrift 99: 1113-1118, 1974.

(81) MEBERG, A., SANDE, H., FOSS, O. P., STENWIG, J. T. Smoking during pregnancy—effects on the fetus and on thiocyanate levels in mother and baby. Acta Paediatrica Scandinavica. 68: 547-552, 1979.

(82) MEYER, M. B. Effects of maternal smoking and altitude on birth weight and gestation. In: Reed, D. M., Stanley, F. J. (Editors). The Epidemiology of Prematurity. Baltimore, Urban and Schwarzenberg, 1977, pp. 81-101.

(83) MEYER, M. B. How does maternal smoking affect birth weight and maternal weight gain? Evidence from the Ontario Perinatal Mortality Study. American Journal of Obstetrics and Gynecology 131(8): 888-893, August 15, 1978.

(84) MEYER, M. B. Reply to Rush. American Journal of Obstetrics and Gynecology 135(2): 282-284, September 1979.

(85) MEYER, M. B., COMSTOCK, G. W. Maternal cigarette smoking and perinatal mortality. American Journal of Epidemiology 96(1): 1-10, July 1972.

(86) MEYER, M. B., JONAS, B. S., TONASCIA, J. A. Perinatal events associated with maternal smoking during pregnancy. American Journal of Epidemiology 103(5): 464-476, 1976.

(87) MEYER, M. B., TONASCIA, J. A. Maternal smoking, pregnancy complications, and perinatal mortality. American Journal of Obstetrics and Gynecology 128(5): 494-502, July 1, 1977.

(88) MEYER, M. B., TONASCIA, J. A., BUCK, C. The interrelationship of maternal smoking and increased perinatal mortality with other risk factors. Further analysis of the Ontario Perinatal Mortality Study, 1960-1961. American Journal of Epidemiology 100(6): 443-452, 1975.

(89) MILLER, H. C., HASSANEIN, K. Maternal smoking and fetal growth of full term infants. Pediatric Research 8: 960-963, 1964.

(90) MILLER, H. C., HASSANEIN, K., HENSLEIGH, P. A. Fetal growth retardation in relation to maternal smoking and weight gain in pregnancy. American Journal of Obstetrics and Gynecology 125(1): 55-60, May 1, 1976.

(91) MULCAHY, R., MURPHY, J., MARTIN, F. Placental changes and maternal weight in smoking and nonsmoking mothers. American Journal of Obstetrics and Gynecology 106(5): 703-704, March 1, 1970.

(92) MURPHY, J., MULCAHY, R. Cigarette smoking and spontaneous abortion. (Letter) British Medical Journal 1(6618): 988, April 15, 1978.

(93) NAEYE, R. Effects of maternal cigarette smoking on the fetus and placenta. British Journal of Obstetrics and Gynecology 85: 732-737, October 1978.

(94) NAEYE, R. L. Causes of perinatal mortality in the U.S. Collaborative Perinatal Project. Journal of the American Medical Association 238(3): 228-229, July 18, 1977.

(95) NAEYE, R. L., HARKNESS, W. L., UTTS, J. Abruptio placentae and perinatal death: a prospective study. American Journal of Obstetrics and Gynecology 128(7): 740-746, August, 1, 1977.

(96) NAEYE, R. L., LADIS, B., DRAGE, J. S. Sudden infant death syn-

drome: a prospective study. American Journal of Diseases in Children 130: 1207-1210, November 1976.

(97) NEBERT, D. W., WINKER, J., GELBOIN, H. V. Aryl hydrocarbon hydroxylase activity in human placenta from cigarette smoking and nonsmoking women. Cancer Research 29: 1763-1769, October 1969.

(98) NERI, A., ECKERLING, B. Influence of smoking and adrenaline (epinephrine) on the uterotubal insuflation test (Rubin test). Fertility and Sterility 20(5): 818-828, 1969.

(99) NEUTEL, C. I., BUCK, C. Effect of smoking during pregnancy on the risk of cancer in children. Journal of the National Cancer Institute 47(1): 59-63, July 1971.

(100) NEWBY, M. B., ROBERTS, R. J., BHATNAGAR, R. K. Carbon monoxide—hypoxia-induced effects on catecholamines in the mature and developing rat brain. Journal Pharmacology and Experimental Therapeutics 206: 61-68, 1978.

(101) NIKONOVA, T. V. Transplacental action of benzo(a)pyrene and pyrene. Bulletin of Experimental Biology and Medicine 84: 1025-1027, 1977.

(102) NISWANDER, J. R., GORDON, M. (Editors). Maternal characteristics. Section 1. Demographic characterics. Cigarette smoking. In: The Women and Their Pregnancies. The Collaborative Perinatal Study of the National Institute of Neurological Diseases and Stroke. DHEW Publication No. (NIH) 73-379, 1972.

(103) OBE, G., HERHA, J. Chromosomal aberrations in heavy smokers. Human Genetics 41: 259-263, 1978.

(104) O'LANE, J. M. Some fetal effects of maternal cigarette smoking. Obstetrics and Gynecology 22(2): 181-184, August 1963.

(105) OLUBADEWO, J. O., SASTRY, B.V.R. Human placental cholinergic system: stimulation-secretion coupling for release of acetylcholine from isolated placental villus. Journal of Pharmacology and Experimental Therapeutics 204: 433-455, 1978.

(106) ONTARIO DEPARTMENT OF HEALTH. Second Report of the Perinatal Mortality Study in Ten University Teaching Hospitals. Toronto, Canada, Ontario Department of Health, Ontario Perinatal Mortality Study Committee, Volume I, 1967, 275 pp.

(107) ONTARIO DEPARTMENT OF HEALTH. Supplemental to the Second Report of the Perinatal Mortality Study in Ten University Teaching Hospitals. Toronto, Canada, Ontario Department of Health, Ontario Perinatal Mortality Study Committee, Volume II, 1967, pp. 95-275.

(108) OSCHNER, A. Cigarette smoking: principal factor that accelerates aging in man. Journal of the American Geriatric Society 24: 385-393, 1976.

(109) PALMGREN, B., WAHLEN, T., WALLANDER, B. Toxaemia and cigarette smoking during pregnancy. Prospective consecutive investigation of 3927 pregnancies. Acta Obstetrica et Gynecologica Scandinavica 52: 183-185, 1973.

(110) PALMGREN, B., WALLANDER, B. Cigarettrokning och abort. Konsekutiv prospektiv undersokning av 4312 graviditeter (Cigarette smoking and abortion. Consecutive prospective study of 4,312 pregnancies). Lakartidningen 68(22): 2611-2616, May 26, 1971.

(111) PELKONEN, O., JOUPPILA, P., KARKI, N. T. Effect of maternal cigarette smoking on 3,4-benzyprene and n-methylaniline metabolism in human fetal liver and placenta. Toxicology and Applied Pharmacology 23: 399-407, 1972.

(112) PELKONEN, O., KARKI, N. T., KOIVISTO, M., TUIMALA, R., KAUPPILA, A. Maternal cigarette smoking, placenta aryl hydrocarbon

hydroxylase and neonatal size. Toxicological Letters 3: 331-335, 1979.

(113) PERSKY, H., O'BRIEN, C. P., FINE, E., HOWARD, W. J., KAHN, M. A., BECK, R. W. The effect of alcohol and smoking on testicular function and agression in chronic alcoholics. American Journal of Psychiatry 134: 621-625, 1977.

(114) PERSSON, P. H., GRENNERT, L., GENNSER, G., KULLANDERS, S. A study of smoking and pregnancy with special reference to fetal growth. Acta Obstetrica et Gynecologica Scandinavica, Supplement 78: 33-39, 1978.

(115) PETTERSSON, F., FRIES, H., NILLIUS, S. J. Epidemiology of secondary amenorrhea. I. Incidence and prevalence rates. American Journal of Obstetrics and Gynecology 117: 80-86, 1973.

(116) PETTIGREW, A. R., LOGAN, R. W., WILLOCKS, J. Smoking in pregnancy—effects on birth weight and on cyanide and thiocyanate levels in mother and baby. British Journal of Obstetrics and Gynecology 84: 31-34, 1977.

(117) PIRANI, B. B. K., MACGILLIVRAY, I. Smoking during pregnancy: its effects on maternal metabolism and fetoplacental function. American Journal of Obstetrics and Gynecology 52: 257-263, 1978.

(118) QUIGLEY, M. E., SHEEHAN, K. L., WILKES, M. M. AND YEN, S. S. C. Effects of maternal smoking on circulating catecholamine levels and fetal heart rates. American Journal of Obstetrics and Gynecology 133: 685-690, 1979.

(119) RANTAKALLIO, P. Relationship of maternal smoking to morbidity and mortality of the child up to the age of five. Acta Paediatrica Scandinavica 67: 621-631, 1978.

(120) RANTAKALLIO, P. The effect of maternal smoking on birth weight and the subsequent health of the child. Early Human Development 2(4): 371-382, December 1978.

(121) RANTAKALLIO, P., KRAUSE, U., KRAUSE, K. The use of ophthalmological services during the preschool age, ocular findings and family background. Journal of Pediatric Ophthalmology and Strabismus 15(4): 253-258, July/August 1979.

(122) RESNIK, R., BRINK, G. W., WILKES, M. Catecholamine-mediated reduction in uterine blood flow after nicotine infusion in the pregnant ewe. Journal of Clinical Investigation 63: 1113-1136, 1979.

(123) RHEAD, W. J. Smoking and SIDS. Pediatrics 59(5): 791-792. May 1977.

(124) ROELS, H., HUBERMONT, G., BUCHET, J. P., LAUWERYS, R. Placental transfer of lead, mercury, cadmium, and carbon monoxide in women: III. Factors influencing the accumulation of heavy metals in the placenta and the relationship between metal concentration in the placenta and in maternal and cord blood. Environmental Research 16: 236-247, 1978.

(125) ROWELL, P. P., SASTRY, B. V. R. Human placental cholinergic system: effects of cholinergic blockade on amino acid uptake in isolated placental villi. Federation Proceedings 36: 981, 1977.

(126) ROWELL, P. P., SASTRY, B. V. R. The influence of cholinergic blockade on the uptake of amino-isobutyric acid by isolated human placental villi. Toxicology Applied Pharmacology 45: 79-93, 1978.

(127) RUSH, D. Effects of smoking on pregnancy and newborn infants. American Journal of Obstetrics and Gynecology 135(2): 281-282, September 1979.

(128) RUSH, D., KASS, E. H. Maternal smoking: a reassessment of the association with perinatal mortality. American Journal of Epidemiology 96 (3): 183-196, September 1972.

(129) RUSSELL, C. S., TAYLOR, R., LAW, C. E. Smoking in pregnancy, maternal blood pressure, pregnancy outcome, baby weight and growth, and other related factors. A prospective study. British Journal of Preventive and Social Medicine 22(30): 119-126, July 1968.

(130) RUSSELL, C. S., TAYLOR, R., MADDISON, R. N. Some effects of smoking in pregnancy. Journal of Obstetrics and Gynaecology of the British Commonwealth 73: 742-746, October 1966.

(131) SASTRY, B. V. R., OLUBADEWO, J. O., BOEHM, F. H. Effects of nicotine and cocaine on the release of acetylcholine from isolated human placental villi. Archives Internationales Pharmacodynamie et de Therapie 229: 23-36, 1977.

(132) SAXTON, D. W. The behavior of infants whose mothers smoke in pregnancy. Early Human Development 2/4: 363-369, 1978.

(133) SCHIRREN, C., GEY, G. Der Einflusz des Rauchens auf de Fortpflanzungs-fahigkeit beir Mann und Fran. Zeitschrift fur Haut und Geschlechts—Krankheiten 44: 175-182, 1969.

(134) SCHLEDE, E., MERKER, H. J. Effect of benzo(a)-pyrene treatment on the benzo(a)pyrene hydroxylase activity in maternal liver, placenta, and fetus of the rat during Day 13 to Day 18 of gestation. Naunyn-Schmiedeberg's Archives of Pharmacology 272(1): 89-100, December 21, 1972.

(135) SCHORAH, C. J., ZEMROCH, P. J., SHEPPARD, S., SMITHELLS, R. W. Leucocyte ascorbic acid and pregnancy. British Journal of Nutrition 39: 139-149, 1978.

(136) SCHWARTZ, D., GOUJARD, J., KAMINSKI, M., RUMEAU-ROUQUETTE, C. Smoking and pregnancy: results of a prospective study of 6,989 women. Revue Europeene d'Etudes Cliniques et Biologiques 17 (9): 867-879, 1972.

(137) SCHWETZ, B. A., SMITH, F. A., LEONG, B. K. J., STAPLES, R. E. Teratogenic potential of inhaled carbon monoxide in mice and rabbits. Teratology 19: 385-392, 1979.

(138) SIMPSON, W. J. A preliminary report on cigarette smoking and the incidence of prematurity. American Journal of Obstetrics and Gynecology 73(4): 808-815, April 1957.

(139) STEELE, R., LANGWORTH, J. T. The relationship of antenatal and postnatal factors to sudden unexpected death in infancy. Canadian Medical Association Journal 94: 1165-1171, May 28, 1966.

(140) STOA, K. F. Studies on thiocyanate in serum. In: Second Medical Yearbook, Bergen, Norway, University of Bergen, 1957, 14 pp.

(141) SUZUKI, K., HORIGUCHI, T., COMAS-URRUTIA, A. C., MUELLER-HEUBACH, E., MORISHIMA, H. O., ADAMSONS, K. Pharmacologic effects of nicotine upon the fetus and mother in the rhesus monkey. American Journal of Obstetrics and Gynecology 111(8): 1092-1101, December 15, 1971.

(142) SUZUKI, K., HORIGUCHI, T., COMAS-URRUTIA, A. C., MUELLER-HEUBACH, E., MORISHIMA, H. O., ADAMSONS, K. Placental transfer and distribution of nicotine in the pregnant rhesus monkey. American Journal of Obstetrics and Gynecology 119(2): 253-262, May 15, 1974.

(143) TOKUHATA, G. Smoking in relation to infertility and fetal loss. Archives of Environmental Health 17: 353-359, 1968.

(144) UNDERWOOD, P., HESTER, L. L., LAFFITTE, T., JR., GREGG, K. V. The relationship of smoking to the outcome of pregnancy. American Journal of Obstetrics and Gynecology 91(2): 270-276, January 15, 1965.

(145) UNDERWOOD, P. B., KESLER, K. F., O'LANE, J. M., CALLAGAN, D.

A. Parental smoking empirically related to pregnancy outcome. Obstetrics and Gynecology 29(1): 1-8 January, 1967.

(146) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE Smoking and Health: A Report of the Surgeon General. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health. DHEW Publication No. (PHS) 79-50066, 1979, 1251 pp.

(147) U.S. PUBLIC HEALTH SERVICE. The Health Consequences of Smoking, 1973. U.S. Department of Health, Education, and Welfare, DHEW Publication No. (HSM) 73-8704, 1973, pp. 99-149.

(148) VAUGHT, J. B., GURTOO, H. L., PARKER, N. B., LEBOEUF, R., DOCTOR, G. Effects of smoking on benzo(a)pyrene metabolism by human placental microsomes. Cancer Research 39(8): 3177-3183, August 1979.

(149) VESSEY, M. P., WRIGHT, N. H., MCPHERSON, K., WIGGINS, P. Fertility after stopping different methods of contraception. British Medical Journal 1 (6108): 265-267, 1978.

(150) VICZIAN, M. Results of spermatozoa studies in cigarette smokers. Zeitschrift fur Haut und Geschlechts-Krankheiten 44(5): 183-187, 1969.

16*(151)*VICZIAN, M. The effect of cigarette smoke inhalation on spermatogenesis in rats. Experientia 24: 511-513, 1968.

(152) WEBSTER, W. SV. Cadmium-induced fetal growth retardation in the mouse. Archives of Environmental Health 33(1): 36-42, January/February 1978.

(153) WELCH, R. M. GOMMI, B., ALVARES, A. P., CONNEY, A. H. Effect of enzyme induction on the metabolism of benzo(a)pyrene and 3-methyl-4-monomethylaminoazobenzene in the pregnant and fetal rat. Cancer Research 32(5): 973-978, May 1972.

(154) WENNERBERG, P. A., WELSCH, F. Effects of cholinergic drugs on uptake of 14 C-aminoisobutyric acid by human term placenta fragments: implication for acetylcholine recognition sites and observations on the binding of radioactive cholinergic ligands. Federation Proceedings 36: 980, 1977.

(155) WILSON, E. W. The effect of smoking in pregnancy on the placental co-efficient. New Zealand Medical Journal 74(475): 384-385, 1972.

(156) WINGERD, J., CHRISTIANSON, R., LOVITT, W. V., SCHODEN, E. J. Placental ratio in white and black women: relation to smoking and anemia. American Journal of Obstetrics and and Gynecology 124(7): 671-675, April 1, 1976.

(157) WINGERD, J., SCHOEN, E. J. Factors influencing length at birth and height at five years. Pediatrics 53(5): 737-741, May 1974.

(158) WINTERNITZ, W. W., QUILLEN, D. Acute hormonal response to cigarette smoking. Journal Clinical Pharmacology 17(7): 389-397, July 1977.

(159) YERUSHALMY, J. Mother's cigarette smoking and survival of infant. American Journal of Obstetrics and Gynecology 88(4): 505-518, February 15, 1964.

(160) YERUSHALMY, J. The relationship of parents' cigarette smoking to outcome of pregnancy—implications as to the problem of inferring causation from observed associations. American Journal of Epidemiology 93(6): 443-456, June 1971.

(161) YOSHINAGA, K., RICE, C., KRENN, J., PILOT, R. L. Effects of nicotine on early pregnancy in the rat. Biology of Reproduction 20: 294-303, March, 1979.

(162) **ZABRISKIE, J. R.** Effect of cigarette smoking during pregnancy. Study of 2000 cases. Obstetrics and Gynecology 21(4): 405-411, April 1963.

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PEPTIC ULCER DISEASE

There is little information dealing specifically with the relationship between smoking and peptic ulcer disease in women. The data which are available suggest the same trend toward higher prevalence of peptic ulcer disease among women who smoke as is observed among men who smoke. Table 1, extracted from the 1979 Surgeon General's Report, shows that the prevalence of "peptic ulcer" in female smokers was higher in two out of three studies of women, which showed a twofold or 1.6 fold higher prevalence (7). The one study which failed to demonstrate an increased prevalence was conducted in rural Poland where very few women smoke (only 7 percent) (6). The median ratio of smoking ulcer patients to nonsmoking ulcer patients has been reported to be 1.7 for men (7). Thus, women smokers seem to show greater susceptibility to ulcer disease than do nonsmokers.

The population of women with ulcers contains a greater proportion of smokers than does the group of women without ulcers. Alp et al. performed a retrospective analysis of 638 patients with gastric ulcer, 230 of whom were women (2). There were 1.9 times as many smokers in the group of women ulcer patients as in an age-matched control group. However, even among the ulcer patients, only 39 percent were smokers. In a smaller series of 31 female patients admitted to hospitals with hemorrhage from, or perforation of, gastric or duodenal ulcers, the prevalence of smoking was 26 percent in both ulcer patients (8/31) and controls (8/31) (1).

In a report examining the effect of smoking on healing rates of gastric and duodenal ulcers, Doll et al. studied 92 women with gastric ulcer and 54 women with duodenal ulcer (3). Smoking was 1.6 times more common in women gastric ulcer patients as in controls matched for age and place of residence ($p < 0.01$). There was no significant excess in the proportion of smokers in the group with duodenal ulcer. The effect of smoking on healing rate was reported for men and women grouped together, so no conclusion regarding specific effects on women is possible.

Although some studies of etiological factors in smoking-induced ulcer disease (gastric acid secretion, pancreatic secretion, etc.) have included women, the number of women has been small, or the data from women have not been presented separately.

In summary, the evidence currently available documents an increased prevalence of peptic ulcer disease in women who smoke. No data are available concerning specific effects of smoking in women on gastric acid secretion, gastric emptying,

TABLE 1.—Prevalence of peptic ulcer in smoking and nonsmoking women (number per 100)

Reference	No. with ulcers	Smokers	Nonsmokers	Ratio*
Higgins, M.W. (1966) (5)	47	2.8	1.4	2.0
Friedman, G.D. (1974) (4)	1092	6.3	3.9	1.6
Jedrychowski, W. (1974) (6)	26	0.8	1.3	0.6

$$\text{*Ratio} = \frac{\text{Prevalence among smokers}}{\text{Prevalence among nonsmokers}}$$

pancreatic secretion, or other processes which might be involved in the pathogenesis of peptic ulcer disease.

Summary

The 1979 Surgeon General's Report included evidence that cigarette smoking in males was significantly associated with the incidence of peptic ulcer diease and increased the risk of dying from peptic ulcer disease by approximately two-fold. The effect of smoking on pancreatic secretion and pyloric reflux demonstrated among men may provide a mechanism by which peptic ulcers develop.

1. Female smokers show a prevalence of peptic ulcer higher than that of nonsmokers by approximately two-fold.
2. The effect of cessation on healing is not known.

References

(1) ALLIBONE, A., FLINT, F. J. Bronchitis, aspirin, smoking and other factors in the aetiology of peptic ulcer. Lancet 2: 179-182, July 26, 1958.

(2) ALP, M. H., HISLOP, I. G., GRANT, A. K. Gastric ulcer in south Australia 1954-1963. 1. Epidemiological factors. Medical Journal of Australia 2: 1128-1132, December 12, 1970.

(3) DOLL, R., JONES, F. A., PYGOTT, F. Effect of smoking on the production and maintenance of gastric and duodenal ulcers. Lancet 1: 657-662, March 29, 1958.

(4) FRIEDMAN, G. D., SIEGELAUR, A. B., SELTZER, C. C. Cigarettes, alcohol, coffee and peptic ulcer. New England Journal of Medicine 290(9): 469-473, February 28, 1974.

(5) HIGGINS, M. W., KJELSBERG, M. Characteristics of smokers in Tecumseh, Michigan. II. The distribution of selected physical measurements and physiological variables and the prevalence of certain

diseases in smokers and nonsmokers. American Journal of Epidemiology 86: 60-77, 1967.

(6) JEDRYCHOWSKI, W., POPIELA, T. Association between the occurrence of peptic ulcers and tobacco smoking. Public Health London 88(4): 195-200, 1974.

(7) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE. Smoking and Health: a Report of the Surgeon General. Department of Health, Education, and Welfare, Public Health Services, Office of the Assistant Secretary for Health, Office on Smoking and Health. DHEW Publication No. PHS) 79-50066, 1979, 1251 pp.

INTERACTIONS OF SMOKING WITH DRUGS, FOOD CONSTITUENTS, AND RESPONSES TO DIAGNOSTIC TESTS

Since most published studies investigating the effect of cigarette smoking on measures of health were performed in mixed populations, it is difficult to demonstrate specific factors applicable only to women. Neither the differences between men and women regarding the metabolism and action of drugs nor the pharmacological basis for differences between smokers and nonsmokers is well understood. The same is also true of the observed variations in laboratory values and nutritional needs. Thus, the associations for women between smoking, drugs, variations in clinical laboratory values, and nutritional needs require further study.

Women Smokers and Nonsmokers and Drug Consumption Patterns

The drug consumption pattern of women as compared to men has been studied by a number of investigators using different methodologies. The results consistently show that women are prescribed and take more prescription drugs than men (7,17). In one study where 1-year drug histories were used, the percentage of women using prescription drugs was 29 percent as compared to 13 percent for men (17). Another study which examined only drugs consumed within 48 hours of the interview showed that 60.2 percent of the women had taken medication compared to 41.8 percent of the men (7). The two studies cited are unique in the realm of drug usage studies because they measure actual self-administration of drugs rather than counting physician prescriptions or pharmacy dispensing patterns. Unfortunately, neither of these studies quantified information according to whether the subjects were smokers or nonsmokers.

Other reports show that smokers tend to use more drugs, especially of the psychotherapeutic type and drink more coffee and alcoholic beverages than nonsmokers (18,26). In only one study have women smokers and nonsmokers been compared for use of all drug categories; these data were derived from a self-administered questionnaire asking about drug use for the past year (21). As Table 1 shows, women smokers take more of almost every type of drug than nonsmokers. When the data were organized according to age groups, the 15-to-19-year-old group of women showed a marked elevation in drug use among smokers (Table 2).

Although the data are preliminary, a trend that female smokers consume drugs with greater frequency than female nonsmokers is suggested. It is beyond the scope of this chapter

TABLE 1.—Ratio of percent usage of drug classes, women smoker/nonsmoker status

Drug class	White	Black	Asian
Antihistamine or allergy medicine	0.8	0.9	0.6
Cough medicine	1.7	1.8	0.7
Asthma medicine	0.9	1.0	0.9
Aspirin-containing drugs	1.2	1.2	0.9
Pain medicine	1.2	1.2	1.0
Codeine, morphine, Darvon, Percodan, Demerol	1.5	1.6	1.2
Phenobarbital or other barbiturates	1.3	1.8	1.6
Sleeping pills	1.2	1.3	1.3
Tranquilizers	1.5	1.6	1.8
Anticoagulants	1.3	0.8	0.0
Digitalis or other heart medication	1.0	0.8	0.1
Antihypertensives	0.8	1.1	0.9
Diuretics	1.1	1.0	1.3
Cortisone-type medication	1.0	1.2	1.0
Hormones	1.2	1.3	1.4
Insulin or diabetic pills	0.9	0.8	0.9
Iron or anemia medications	0.9	0.9	0.9
Thyroid medication	1.1	1.3	2.3
Pills to control periods	1.3	1.2	1.5
Contraceptives	1.2	1.1	1.3
Benzedrine or Dexedrine	1.6	1.1	1.1
Weight reduction medication	1.1	0.9	1.3
Penicillin or other antibiotics	1.2	1.2	1.0
Sulfa drugs	1.1	1.2	0.8
Stomach or digestion medicine	1.2	1.2	1.3

SOURCE: Seltzer, C.G. (21).

TABLE 2.—Percentage of positive responses among females in age group 15–19

Question	Smokers	Nonsmokers
Taken phenobarbital or barbiturates?	2.3	1.0
Taken codeine, morphine, etc.?	16.0	6.5
Taken Benzedrine or Dexedrine?	4.9	0.3
Taken penicillin or other antibiotics?	33.0	25.8
Taken pills to prevent pregnancy?	27.0	9.7

SOURCE: Seltzer, C.G. (21).

to differentiate between the behavioral components of this phenomenon or to address the argument that women who smoke are less healthy than nonsmokers. It is beneficial, however, to examine the few reports that address the differences in drug action between smokers and nonsmokers, regardless of the reasons for drug use.

Altered Clinical Response to Drug Therapy by Smokers Compared to Nonsmokers

The number of studies investigating the differences in the clinical responses to a drug by smokers and nonsmokers are far fewer in number than the studies examining the alterations in metabolism and biochemistry of drugs in smokers. The 1979 Surgeon General's Report included an extensive review of the alterations in drug disposition that occur in smokers (25). That information is useful for clarifying mechanisms by which smoking alters drug metabolism, absorption, excretion, and other functions. The clinical significance of these alterations has not been clarified, however.

The most exhaustive examination of alterations in smokers' clinical response to drugs was done by Jick and his associates in the Boston Collaborative Drug Surveillance Program (BCDSP). Over the past several years, this group has investigated the clinical response of smokers and nonsmokers to six different drugs: propoxyphene (Darvon) (4); diazepam (Valium) (3); chlordiazepoxide (Librium) (3); phenobarbital (3); chlorpromazine (Thorazine) (24); and theophylline tea (19). The differences observed between smokers and nonsmokers were consistent among men and women, except for the theophylline study, in which the toxic effects of therapy were slightly more frequent among women (13.4 percent) than among men (9.19 percent). Only in the chlorpromazine study (24) did the study group (those taking chlorpromazine) contain more women than men, an observation that supports other reports that women use major tranquilizing agents more frequently than men (18).

Since the published BCDSP data is not organized according to groups of women smokers and nonsmokers, any difference in drug use between these groups is not reflected in the data analysis. However, it is important to note that these studies, except as noted in the chlorpromazine study, predominantly involved men. It has been shown that women report more frequent use of the minor tranquilizers such as diazepam and chlordiazepoxide (17). Thus these studies should not be interpreted as reflecting drug response among the general population (17).

The studies on chlorpromazine, diazepam, and chlordiazepoxide showed a lessened frequency of the adverse effect of drowsiness among smokers as compared to nonsmokers (4,24). Conversely, no difference was reported for phenobarbital (3).

The analgesic effect of propoxyphene was reduced in smokers, an effect which was not observed in smokers on aspirin, codeine, acetaminophen, or combinations of these drugs (4).

The evidence for increased theophylline metabolism in smokers is well established and predicts the observed clinical response to theophylline (13). The BCDSP study of theophylline showed that smokers not only required larger doses of theophylline for efficacy, but also were less likely to report adverse effects than nonsmokers, even though they required larger doses.

Theoretically, then, because of a decreased clinical response to a drug, the tendency would be for the smoker to require increased doses to achieve the same therapeutic effect as a nonsmoker.

Therapeutic efficacy and adverse side effects in relationship to gender, smoking history, and drug consumption patterns have not been adequately studied, although the preliminary evidence would indicate an area of potential toxic drug effects and/or therapeutic failures.

Oral Contraceptives and Smoking

Chronic estrogen therapy has a profound interaction with chronic tobacco use. Again, the BCDSP has been most instrumental in assessing the influence of these two factors on the health status of women.

In assessing the relative risk of stroke in women who smoke and take oral contraceptives, the data from the Collaborative Group for the Study of Stroke in Young Women show that smoking alone increased the risk of hemorrhagic stroke (i.e., subarachnoid) from 1.0 for a nonsmoker who did not use oral contraceptives, to 2.6 for a smoker who did not use oral contraceptives. A smoker taking oral contraceptives had a relative risk of 6.1 or 7.6 (depending on the control group) (6). Similar increases in risks do not seem to occur for thrombotic stroke in the smoker taking oral contraceptives, but the risk of a thrombotic stroke for a woman using oral contraceptives alone is about nine times greater than that for a noncontraceptive user (5).

Again using the BCDSP data, the risk of nonfatal myocardial infarction among women under 38 is very low among nonsmokers, whether or not they use oral contraceptives. However, the risk to women who both smoke and use oral contraceptives is substantially higher, ranging from an estimated one per 8,400 annually in women aged 27 to 37 years to one per 250 for women aged 44 to 45 years (16). In a similar study of noncontraceptive estrogens, similar risks were demonstrated for women who both smoke and use estrogens (15). These findings are in agreement with studies done in Great Britain where oral contraceptives were associated with an overall increase in cardiovascular disease in young women (20).

Another group which has investigated the link between smoking, oral contraception, and myocardial infarction reported that there is a considerable interaction between smoking and contraceptive use. The group found that rate of acute myocardial infarction among female smokers on oral contraceptives is greater than could be accounted for by either smoking or contraceptives alone (22). In earlier studies this same group concluded that there was a dose–response relationship between smoking and myocardial infarction in women, and that among women smoking 35 or more cigarettes per day, the rate of myocardial infarction was estimated to be 20 times higher than among those who never smoked (23).

These data lend themselves to the prediction of risk in only a very general way and provide no particular measures by which a woman—smoker or nonsmoker—can evaluate her own risk of experiencing one of the adverse effects described.

The following section reviews some of the laboratory values that are altered by smoking. Unfortunately, many of the largest studies on the correlation between smoking and alterations in clinical laboratory values have focused on men.

Alterations in Normal Clinical Laboratory Values in Women Smokers

Only a few investigators have studied clinical laboratory values in women smokers and nonsmokers (1,8-12,14,27). Many of these studies show statistically significant differences in a variety of common parameters. The clinical significance of these differences may not be apparent, however, since the actual differences between women smokers and nonsmokers are small. For example, a study of packed red cell volume (PCV) and hemoglobin (Hb) in women smokers and nonsmokers showed the PCV and Hb for nonsmokers to be 41.95 and 13.85 compared to 42.94 and 14.16 for smokers, a difference significant at $p < 0.05$, but a discrimination which physician or patient may find difficult to assess (14).

Small differences in laboratory values between smokers and nonsmokers can be seen in a number of serum chemistry and hematologic tests. One measurement that shows a wide enough variation between smokers and nonsmokers to be recognized clinically is the leukocyte count of a smoker (11,12). It is important to recognize that a WBC of 12,000 per mm^3 is within the normal range for a heavy cigarette smoker, and that the differential count remains normal (11). In one study, individuals with chronic bronchitis were excluded from evaluation of leukocyte counts, and the same relative increase in leukocyte count was observed (12).

In several studies of triglyceride and cholesterol values in smoking and nonsmoking women, an elevation of both values, which was not statistically significant, was seen in smokers. The addition of oral contraceptive use to smoking caused a significant elevation over the nonsmoker, noncontraceptive user. The nonsmoker values were 79 ± 6.8 mg/100 ml for triglycerides and 157 ± 7.5 mg/100 ml for cholesterol. In the smoker they were 110 ± 14.8 mg/100 ml and 174.3 ± 8.8 mg/100 ml respectively, whereas the smoker using oral contraceptives had a triglyceride value of 150.0 ± 14.1 mg/100 ml and a cholesterol value of 186.1 ± mg/100 ml. In this same study, there was no significant difference between the levels of vitamins A, E or C in smoking and nonsmoking women (27).

A number of investigators have measured vitamin C levels in smoking and nonsmoking women, with extreme variation in results. Some showed decreased plasma and leukocyte vitamin C levels in smokers, and others showed no differences between smokers and nonsmokers. The discrepancies in these results may in part be related to the amount of dietary vitamin C habitually consumed by the subjects in the various studies (27).

Changes in serum proteins were the subject of another study of women smokers and nonsmokers (26). Significant differences in all serum protein fractions were found in cigarette smokers compared to nonsmokers. In general, the effects increased with the amount smoked. Past smokers showed globulin values that were significantly below those of women who never smoked, but there was no difference observed in the other serum protein fractions between past smokers and those who had never smoked.

The Influence of Smoking on the Nutritional Needs of Women

Outside of a possibly increased need for vitamin C in women who smoke, there is very little information about other nutrient requirements in smokers. In recent years a great deal of time has been spent studying the influence of smoking on fetal development, a subject covered elsewhere in this volume. The special nutritional needs of the nonpregnant smoking woman have not been dealt with in any systematic way.

A recent study involving obese women looked at the influence of smoking cessation on body weight (2). Although the data are innately biased because the study group consisted of women enrolled in a weight loss program, the results showed that women who smoked less than a half pack of cigarettes a day gained 4 pounds after they quit. Heavy smokers consuming over two packs a day gained an average of 30 pounds over several decades. Moderate smokers gained an intermediate amount. This study does not contradict a commonly held notion that women gain weight when they stop smoking; however, it provides no behavioral or physiological hypothesis for this phenomenon.

Summary

Most published studies investigating the effects of cigarette smoking on drug use have been performed on mixed populations; factors specific for women have not been demonstrated to date. It has, however, been clearly demonstrated that women are prescribed and consume more prescription drugs than men.

1. Studies of selected drugs indicate that smoking may affect clinical responses and alter the dose required for an effective therapeutic result.
2. Smoking interacts with oral contraceptive use to increase the risk of myocardial infarction and subarachnoid hemorrhage.
3. Common clinical laboratory parameters are altered in smokers compared to nonsmokers; the health significance of these changes is unknown.
4. Insufficient information exists for assessment of the impact of smoking on the nutritional needs of women.

References

(1) BILLIMORIA, J. D., POZNER, H., METSELAAR, B., BEST, F. W., JAMES, D. C. D. Effect of cigarette smoking on lipids, lipoproteins, blood coagulation, fibrinolysis and cellular components of human blood. Atherosclerosis 21(1): 61-76, January-February 1975.

(2) BLITZER, P. H., RIMM, A. A., GIFFER, E. E. The effect of cessation of smoking on body weight in 57,032 women: cross sectional and longitudinal analysis. Journal of Chronic Diseases 30(7): 415-429, July 1977.

(3) BOSTON COLLABORATIVE DRUG SURVEILLANCE PROGRAM. Clinical depression of the central nervous system due to diazepam and chlordiazepoxide in relation to cigarette smoking and age. New England Journal of Medicine 288(6): 277-280, February 8, 1973.

(4) BOSTON COLLABORATIVE DRUG SURVEILLANCE PROGRAM. Decreased clinical efficacy of propoxyphene in cigarette smokers. Clinical Pharmacology and Therapeutics 14(2): 259-263, March-April 1973.

(5) COLLABORATIVE GROUP FOR THE STUDY OF STROKE IN YOUNG WOMEN. Oral contraceptives and increased risk of cerebral ischemia or thrombosis. New England Journal of Medicine 288(17): 871-878, April 26, 1973.

(6) COLLABORATIVE GROUP FOR THE STUDY OF STROKE IN

YOUNG WOMEN. Oral contraceptives and stroke in young women. Journal of the American Medical Association 231(7): 718-722, February 17, 1975.

(7) CRAIG, T. L., VANNATTA, P. A. Current medication use and symptoms of depression in a general population. American Journal of Psychiatry 135(9): 1036-1039, September 1978.

(8) DALES, L. G., FRIEDMAN, Q. D., SIEGELAUB, A. B., SELTZER, A. C. Cigarette smoking and serum chemistry tests. Journal of Chronic Diseases 27(6): 293-307, August 1974.

(9) DALES, L. G., FRIEDMAN, Q. D., SEIGELAUB, A. B., SELTZER, A. C., URY, H. K. Cigarette smoking habits and urine characteristics. Nephron 20: 163-170, 1978.

(10) DESMOND, P. V., ROBERTS, R. K., WILKINSON, Q. R., SCHENKER, S. No effect of smoking on metabolism of chlordiazepoxide. New England Journal of Medicine 300(4): 199-200 January 25, 1979.

(11) FRIEDMAN, Q. D., SIEGELAUB, A. B., SELTZER, C. C., FELDMAN, R., COLLEN, M. F. Smoking habits and the leukocyte count. Archives of Environmental Health 26(3): 137-143. March 1973.

(12) HELMAN, N., RUBENSTEIN, L. S. The effects of age, sex, and smoking on erythrocytes and leukocytes. American Journal of Clinical Pathology 63: 35-44, 1975.

(13) HUNT, S. N., JUSKO, W. J., YURCHAK, A. M. Effect of smoking on theophylline disposition. Clinical Pharmacology and Therapeutics 19(5, Part 1): 546-551, May 1976.

(14) ISAGER, H., HAGERUP, L. Relationship between cigarette smoking and high packed cell volume and haemoglobin levels. Scandinavian Journal of Haemotology 8(4): 241-244, 1971.

(15) JICK, H., DINAN, B., ROTHMAN, K. J. Noncontraceptive estrogens and nonfatal myocardial infarction. Journal of the American Medical Association 239(14): 1407-1408, April 3, 1978.

(16) JICK, H., DINAN, B., ROTHMAN, K. J. Oral contraceptives and nonfatal myocardial infarction. Journal of the American Medical Association 239(14): 1403-1406, April 3, 1978.

(17) PARRY, H. F., BALTER, M. B., MELLINGER, Q. D., CISIN, I. H., MANHEIMER, D. I. National patterns of psychotherapeutic drug use. Archives of General Psychiatry 28: 769-783, June 1973.

(18) PARRY, H. F., CISIN, I. H., BALTER, M. B., MELLINGER, Q. D., MANHEIMER, D. I. Increasing alcohol intake as a coping mechanism for psychic stress. In: Cooperstock, R. (Editor). Social Aspects and Medical Use of Psychotropic Drugs. Toronto, Addiction Research Foundation, 1974.

(19) PFEIFER, H. J., GREENBLATT, D. J. Clinical toxicity of theophylline in relation to cigarette smoking. Chest 73(4): 455-459, April 1978.

(20) ROYAL COLLEGE OF GENERAL PRACTITIONERS ORAL CONTRACEPTION STUDY. Mortality among oral contraceptive users. Lancet (4): 727-733, October 8, 1977.

(21) SELTZER, C. G., FRIEDMAN, Q. D., SIEGELAUB, A. B. Smoking and drug consumption in white, black, and oriental men and women. American Journal of Public Health 64(5): 466-473, March 1974.

(22) SHAPIRO, S., SLONE, D., ROSENBERG, L., KAUFMAN, D., STOLLEY, P.D., MIETTINEN, O. S. Oral contraceptive use in relation to myocardial infarction. Lancet (1): 743-747, April 7, 1979.

(23) SLONE, D., SHAPIRO, S., ROSENBERG, L., KAUFMAN, D. W., HARTZ, S. C., ROSSI, A. C., STOLLEY, P. D., MIETTINEN O. S. Relation of cigarette smoking to myocardial infarction in young women.

New England Journal of Medicine 298(23): 1273-1276, 1978.

(24) SWETT, D. Drowsiness due to chlorpromazine in relation to cigarette smoking. Archives of General Psychiatry 31: 211-213, August 1974.

(25) U.S. PUBLIC HEALTH SERVICE. Smoking and Health. A Report of the Surgeon General. U.S. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, DHEW Publication No. (PHS) 79-50066, 1979, 1251 pp.

(26) WINGERD, J., SPONZILLI, E. E. Concentrations of serum protein fractions in white women: effects of age, weight, smoking, tonsillectomy and other factors. Clinical Chemistry 23(7): 1310-1317, 1977.

(27) YEUNG, D. L. Relationships between cigarette smoking, oral contraceptives and plasma vitamins A, E, C and plasma triglycerides and cholesterol. American Journal of Clinical Nutrition 29: 1216-1221, 1976.

PSYCHOSOCIAL AND BEHAVIORAL ASPECTS OF SMOKING IN WOMEN

Introduction

Currently, women are rapidly approaching men in the rate of initiation and prevalence of cigarette smoking, but seem to have a lower rate for successful cessation of smoking. (See also Part I of this report, Patterns of Cigarette Smoking.) While an increasing percentage of the U.S. population is giving up smoking, nationwide surveys and cessation studies suggest that a smaller proportion of women than men are quitting successfully.

This part discusses tobacco use by women, with comparative reference to men's use wherever appropriate. Special attention is directed to the patterns of initiation, the rise in smoking among girls, and the factors important in the maintenance of smoking behavior, including pharmacological effects, smoking patterns, information dissemination, and stress management. The differences in successful quitting between men and women smokers are discussed with the hope of generating new ideas for research and intervention.

A separate analysis of smoking patterns among women in the health professions is presented. In addition, a section is devoted to the pregnant smoker because the impact of smoking, both on the fetus and on the pregnant woman, makes this a period of particular importance in the life of the women smoker.

Initiation of Smoking in Adolescent Girls

Cigarette smoking, particularly cigarette smoking among young girls, is a changing phenomenon. Shifts in smoking attitudes and behaviors reflect broader social forces, including changes in sex roles and gender differences in responses to public information programs and to social sanctions against smoking.

The trend in adolescent smoking, as in other "adult-like" behaviors such as alcohol use or sexual activity, is toward earlier onset. For example, before the mid-1970s, girls were less likely to start smoking than boys, and when they did, they started later. Neither of these differences holds true any longer.

A number of psychosocial variables correlate highly with adolescent smoking trends. These include the attitudes, perceptions, and behaviors of adolescent girls, their social setting (family, peer groups) and those broad demographic factors (race, education, family income, urbanicity) that help to define an individual's position within the society.

CONCEPTS OF ADOLESCENT BEHAVIOR

Discussions of adolescence with its attendant problems have seldom differentiated between boys and girls, and no theory or model of adolescent behavior has been developed specifically for girls. However, gender differences in development, cognitive processes, sex-role acquisition and achievement have recently been examined and a number of psychological differences have been identified (24,26,51,68,98,211).

The essence of adolescence is growth, transition, and change. The rate of physical growth in adolescence is more rapid than at any other stage of development except the neonatal stage. Adolescent development is a complicated process which involves increasing self-awareness, intellectual and emotional growth, and physiological changes.

What adults characterize as risk taking in adolescence may be exploration of the limits of identity and capability. Adolescents are attempting to resolve the competing and conflicting demands stemming from childhood experience on the one hand and expectations of adulthood on the other: dependency and compliance versus autonomy and independent decision-making; orientation toward family versus orientation toward peers. They face increasing demands for social and cognitive achievement and for developing the self-control required to handle new psychological, physical, and social situations. Inadequate experience with these challenges or failure to meet them may result in low self-esteem and increased anxiety and stress.

Numerous formulations contributing to a general model of adolescent development have emerged. These include life-span theory and cohort change (52,131), adolescent sexuality (32), and differences between early and late adolescence (85).

Douvan and Adelson have identified issues that distinguish adolescence: for girls they are sexuality, interpersonal-intimacy, and identity issues; for boys they are sexuality, autonomy–assertion–independence and identity issues (51). In this study, conducted in the 1950s, girls evidenced conflict between the social roles for which they were preparing (further education and careers) and the future role they desired (marriage–motherhood). La Farge described a similar female adolescent conflict between social rules and individual perceptions (109). Research published in the 1970s shows that young women still have role conflicts different from those of young men (68).

Research on gender–role differentiation in childhood has provided some insight into developmental differences between girls and boys. Maccoby suggests that these differences may derive from different role models for boys and girls; from the varying responses of significant adults to their behaviors; from biological differences; and from a combination of these (116). Block and Maccoby and Jacklin report that the differences include girls having less confidence in their ability to handle a new task and less sense of control over what happens to them (18,117). Girls also show greater susceptibility to expressed anxiety, greater need for help and reassurance, greater closeness to friends, and more concern for what is socially desirable.

Adolescent behaviors—social or antisocial, adaptive or maladaptive—are a function both of individual choice and of the opportunities for growth and development which a society provides its youth (36). "Not only is the term 'adolescence' a social definition, but what society perceives as an adolescent problem is also socially defined" (52). Similarly, the development of values, motivations, and controls that foster healthy growth and deter the onset of smoking and other undesirable behaviors depends on the opportunities and resources that society makes available to the adolescent.

PREVALENCE AND PATTERNS OF ADOLESCENT CIGARETTE USE

National surveys of adolescent smoking behavior have provided information on gender differences, secular trends, and age subgroupings within the adolescent period. Surveys of smoking patterns, ages 12 to 18, were conducted by the National Clearinghouse for Smoking and Health (NCSH) in 1968, 1970, 1972, and 1974 and by the National Institute of Education (NIE) in 1979 (130,197). Two other periodic surveys, both sponsored by the National Institute on Drug Abuse (NIDA), included cigarette consumption (2,101). A number of studies in specific geographic locales or among specific populations, such as high school students, have also been carried out (198). Differing definitions of a current regular adolescent smoker make comparisons among these studies particularly difficult. In the NCSH and NIE surveys, a regular smoker is defined as one who smokes cigarettes at least weekly. In the NIDA surveys, regular smoking is defined as occurring within the past 30 days.

Prevalence

Table 1 summarizes adolescent cigarette smoking prevalence between 1968 and 1979, by age and gender, as surveyed by NCSH and by NIE. Between 1968 and 1974 there was a significant increase in the percentage of girl smokers in each age category at each point in time, in contrast to the relatively stable prevalence of current regular smoking among boys. A decline in the average age of smoking initiation for both sexes is suggested by the small but significant increase in smoking prevalence among 12 to 14 year olds. (198). Trends in the data from a national study of high school seniors also support the hypothesis of an earlier age of initiation (101).

In the five years from 1974 to 1979, the proportion of 17 to 18 year old girls who smoked changed little, but the proportion of boys who smoked dropped by a third. It was this difference among 17 to 18 year olds that created the overall higher smoking rate for girls as compared with boys in 1979. However, at ages 15 to 16, the drop from 1974 to 1979 was greater for girls than boys, suggesting that the initiation of smoking is also beginning to decline in those girls born after 1962.

The differences in the within-age-group changes in the smoking prevalence of girls may represent an isolated effect on the cohort of girls born in 1963 and 1964. The change was essentially confined to the 15 to 16 year old subgroups who were born during these years. The precise nature of the interaction of social influences on the development and maturation of this cohort is unclear. However, other data suggest that a marked secular change occurred in cigarette smoking attitudes and behavior which was secondary to an increased awareness of the health risks of smoking.

An alternate hypothesis is that the isolated decline in the 15 to 16 year old subgroup may be an artifact produced by the combined trends of reduced initiation of smoking and the initiation at a younger age. Thus, the decline in prevalence among 15 to 16 year old girls would reflect the decreasing percentage of young women who are taking up smoking, but this trend will be masked in the younger age group by the tendency of those girls who are going to take up smoking to do so at a younger age. The 1979 NIE Survey reports that:

> The increasing prevalence of teenage smoking that was observed in the period between 1968 and 1974 has come to a halt, and a decrease in the smoking rates of both boys and girls has taken place. The decrease in boys' smoking was greater than that of girls, resulting in a higher smoking rate for girls than for boys in 1979. Smoking among boys leveled off in the early 1970s, and then began to decrease. It appears that girls are now following this pattern: the smoking rate has leveled off among 17 and 18 year olds, and probably can be expected to decrease over the next few years (130).

Other surveys (Table 2) support these trends in adolescent girls' smoking behavior. Differences between studies in absolute prevalence rates reported are at least partly due to the

TABLE 1.—Estimates of the percentage of current, regular cigarette smokers, adolescents, aged 12 to 18, United States, 1968–1979

Year	Ages 12–14		Ages 15–16		Ages 17–18		Ages 12–18	
	Male	Female	Male	Female	Male	Female	Male	Female
1968	2.9	0.6	17.0	9.6	30.2	18.6	14.7	8.4
1970	5.7	3.0	19.5	14.4	37.3	22.8	18.5	11.9
1972	4.6	2.8	17.8	16.3	30.2	25.3	15.7	13.3
1974	4.2	4.9	18.1	20.2	31.0	25.9	15.8	15.3
1979	3.2	4.3	13.5	11.8	19.3	26.2	10.7	12.7

NOTE: Current regular smoker includes respondent who smokes cigarettes at least weekly.

SOURCE: National Clearinghouse for Smoking and Health (197), National Institute of Education (130).

difference in the definition of a smoker, and differences in survey technique. The National Institute of Education Survey included as current regular smokers both those who smoke one or more cigarettes per week and those who smoke one or more cigarettes a day. The prevalence rates of Abelson, et al. (2) and Johnston, et al. (101) refer to any cigarette smoking in the past 30 days.

The Abelson, et al. data, which were collected 2 years before that of NIE, show the predicted decline, but to a lesser degree (2,130). The Johnston, et al. data suggest that there was an increase in adolescent girls' smoking as measured in samples of high school seniors between 1975 and 1977 (101). Johnston's figures were retrospectively reported and refer only to youngsters born before and during 1960, and therefore, would not be expected to reflect changes occurring in those cohorts born after 1962 where the decline has occurred. This may explain why the Johnston, et al. 1977 sample did not reflect a downturn, and reports of later cohorts of high school seniors should show a stabilization and then a decline in female smoking rates. Results from a study by the same group in 1978 show the predicted downturn in the smoking habits of high school senior girls (from 39.6 percent in 1977 to 38.1 percent in 1978) as well as boys (from 36.6 percent in 1977 to 34.5 percent in 1978) (103).

Age of Initiation of Smoking

The data in Table 1 show that the prevalence of smoking in girls aged 12-14 increased steadily between 1968 and 1974 to a level equal to or slightly higher than boys of the same age. Between 1974 and 1979 the prevalence of smoking stabilized in girls and may have begun to decline. The prevalence of smoking by boys of this age peaked in 1970 and has shown a steady decline since that time. These trends may represent fewer adolescents taking up smoking, with those who do beginning at an earlier age.

Well over one-half of high school seniors—male and female—who smoke regularly, reported first smoking in the ninth grade or earlier (101). It is hard to know whether this earlier onset reflects something specific to cigarette smoking or is attributable to the more general pattern of earlier onset of all "adult-type" behaviors.

This trend toward early initiation of smoking behavior may have a significant impact on the future health of these adolescents, as many of the health risks associated with smoking increase with both earlier onset of smoking and duration of the smoking habit. In addition, the earlier the use of a substance is begun, the longer it is likely to be continued and the more heavily it is likely to be used (26,102,137).

These national surveys do not permit a detailed examination of the initiation process. "Experimenters," those who have smoked at least a few puffs of a cigarette, but not more than 100 cigarettes, are grouped with "never smokers", those who have never taken even a few puffs. "Occasional" smokers are defined as those who smoke less than one cigarette a week but more than 100 cigarettes in a lifetime. Occasional or intermittent smoking is rare among adults. Examining the proportion of "experimenters" at each age and following their subsequent smoking behavior might help clarify the determinants of the initiation process (126).

In one major British study, smoking only a few cigarettes usually led to becoming a regular smoker; only 15 percent of those who smoked more than a single cigarette escaped adoption of smoking as a regular behavior (126). The estimate in this study of 8 percent "occasional smoking" in adolescence is based on a definition of smoking less than daily, but at least one cigarette a week for as long as 1 month. The difference in definition of occasional smoking makes comparison with current U. S. data on adolescents difficult. From 1968 to 1979, the percentage of current occasional smokers (less than once per week) varied between 0.4 percent and 1.6 percent for girls, and 0.4 percent and 2.3 percent for boys (130). McKennell and Thomas estimated that the mean length of time between smoking the first cigarette and adopting regular (daily) smoking was slightly less than 3 years for boys and slightly more than 2 years for girls (126). The difference is probably due to earlier experimentation among boys. The transition from experimental or occasional

TABLE 2.—Percent of adolescents currently using* cigarettes, alcohol and marihuana, by sex: three national surveys compared

Ages 12-18 NIE (1979)				Ages 12-17 Abelson, et al. (1977)				Ages 17-19 High School Seniors Johnson, et al. (1977)			
Ages		1974	1979	Ages		1974	1977	Ages		1975	1977
				Current Cigarette Use							
12-14	F	5.1	4.3	12-13		13	10			—	—
	M	4.2	3.2								
15-16	F	21.6	12.3	14-15		25	22			—	—
	M	18.1	14.6								
17-18	F	26.4	27.0	16-17		38	35			—	—
	M	32.6	19.6								
12-18	F	15.9	13.1	12-17	F	24	22	17-19	F	35.9	39.6
	M	16.3	11.1		M	27	23		M	37.2	36.6
				Current Alcohol Use							
				16-17	F&M	51	52				
				12-17	F	29	25	17-19	F	62.2	65.0
					M	39	37		M	75.0	77.8
				Current Marihuana Use							
				16-17	F&M	20	29				
				12-17	F	11	13	17-19	F	22.5	30.0
					M	12	19		M	32.3	40.7

*NOTE: Definition of current use varies by study. Cigarettes: NIE (1979)—current regular smoker (one or more cigarettes during the past week over and above a minimum five packs) and current occasional smoker (less than one cigarette per week); Abelson, et al. (1977) and Johnston, et al. (1977)—smoked within the past 30 days. Alcohol and marihuana: use within the past month (smokers and nonsmokers).

SOURCE: Abelson, H.I. (2), Johnston, L.D. (101), National Institute of Education (130).

smoking to regular smoking is an extremely important one to study because it may provide a crucial period for intervention before psychosocial or pharmacological dependency is established.

Number of Cigarettes Smoked

In the NCSH/NIE survey (130), a smaller percentage of female smokers than male smokers smoked 10 or more cigarettes per day (61.8 percent versus 73.8 percent in 1974, and 59.0

percent versus 65.6 percent in 1979). The high school senior survey showed male–female rates to be equivalent at the half-pack per day rate, with boys exceeding girls at heavier levels (101). In that study, the proportion of females currently smoking as much as a half-pack per day increased between 1975 and 1977, while the proportion of males smoking at that rate remained constant. The American Cancer Society survey also suggested an increase in the proportion of heavy smokers among adolescent girls compared with stable rates in boys between 1969 and 1975 (216). It reported a fourfold increase in the percentage of female smokers who smoked at least a pack a day, from 10 percent to 39 percent, compared with an unchanged rate of 31 percent among males. The equality in smoking behavior may be extending to the number of cigarettes smoked.

Type of Cigarette Smoked

In adolescent smokers of both sexes, there has been a definite trend toward smoking cigarettes with lower "tar" yields between 1974 and 1979. Figure 1 shows the decline in the "tar" and nicotine levels of the cigarettes smoked by adolescents. Girls appear to be slightly ahead of boys in the use of lower "tar" cigarettes. The trend can be attributed to three factors: the increased marketing of low "tar" cigarettes; the decreased "tar" levels of existing cigarettes; and increased awareness of differential health hazards associated with different kinds of cigarettes (130). It should be noted, however, that the midpoint on the cumulative percentage continuum has dropped only about 1 mg "tar" between 1974 and 1979, from approximately 17.5 mg to approximately 16.5 mg, and the percentage of adolescents smoking the lowest category of "tar" (less than or equal to 10 mg) is still very small.

Smoking Cessation

Are there differences between girls and boys in patterns of smoking cessation comparable to those observed in adults? A greater proportion of adult males than adult females have quit smoking (see the section on adult smoking cessation in this part). Two national surveys have shown more ex-smokers among adolescent boys than among girls (101,130). Looking at either the percentage of ex-smokers among all adolescents or at the quit rates (number of former smokers divided by number of ever smokers), boys exceed girls in every survey between 1968 and 1979 (130). However, if experimental smokers are eliminated from the analysis, there are no differences between the boys and girls. For the two most recent surveys, the quit rates

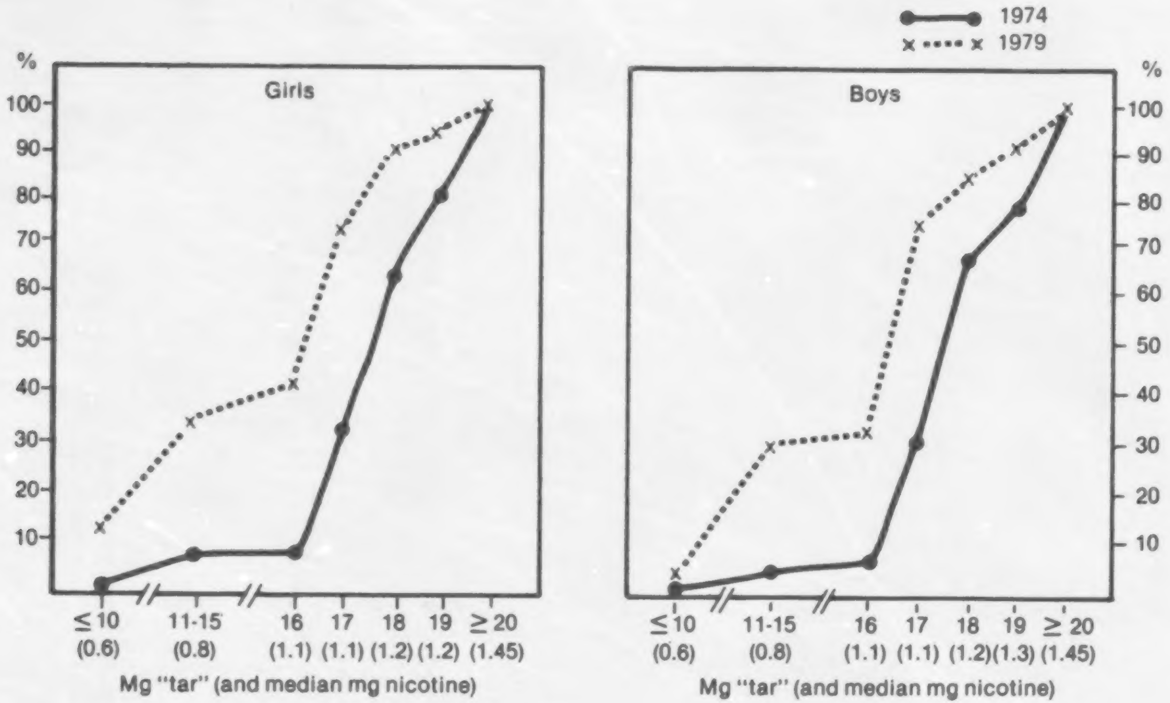

FIGURE 1.—Cumulative percentage of adolescent smokers by the tar level of cigarette smoked, 1974 and 1979

SOURCE: Federal Trade Commission (61), National Institute of Education (130).

were as follows: 33.2 percent of female and 36.0 percent of male smokers had quit in 1974; 30.5 percent of female and 42.3 percent of male smokers had quit in 1979. In contrast, Reeder found no difference in quit rates between boys and girls aged 13 to 19 in national surveys conducted in 1965 (boys 28 percent, girls 29 percent) and in 1975 (boys 34 percent, girls 35 percent) (148). Therefore, it is unclear whether adolescent girls show the same patterns of quitting smoking found in adult women. It should also be remembered that research on both smoking cessation and illicit drug use has shown that quitting is often not a permanent state (100,147,173).

Smoking Prevalence and Ethnicity

There are no data based on a national sample examining adolescent smoking in different racial groups. However, beginning in 1969-1970 Brunswick has conducted a longitudinal personal home interview survey of a representative sample of 668 urban, non-Hispanic black youths in Harlem, New York City. She found that more 16 to 17 year old girls than boys smoked (62 percent versus 50 percent). This was well before national rates had shown smoking among girls equaling and then exceeding that among boys. This greater smoking prevalence in girls continued into the young adult years. The same subjects were reinterviewed 6 to 8 years later, when the youths were aged 18 to 23. Sixty-two percent of young black women (N =258) were current smokers and 18 percent were currently smoking at least a pack a day. This is compared with 57 percent of the black men 18 to 23 years old (N =277) who were current smokers, 16 percent of whom regularly smoked at least a pack a day. These prevalence rates are well above the rates for adult black women found in national survey data, but are only slightly higher than the rates found in adult black men (198). This study is of substantial interest, but may not be representative of national black adolescent smoking patterns.

Alcohol and Marihuana Use

Cigarette use should be viewed in the context of other substance use behaviors. Abelson, et al., provided information on the use of other substances in the age range of 12 to 17 by current cigarette smokers and by those not currently smoking (2). Smokers far exceeded nonsmokers in reporting use of alcohol, marihuana and/or hashish, or "stronger" drugs (hallucinogens, cocaine, heroin, and other opiates): positive replies for alcohol were 80.0 percent versus 44.8 percent; for marihuana and/or hashish, 68.3 percent versus 16.7 percent; and for stronger drugs, 26.3 percent versus 4.1 percent respectively (24, 103,130,216). Similar figures for alcohol use by 13 to 17 year old girls were reported by Yankelovich, et al.: 81 percent of the smokers drank compared with 42 percent of nonsmokers, but somewhat lower estimates were reported for marihuana use—25 percent of the smokers versus 3 percent of the nonsmokers (203). Strong associations between alcohol use and cigarette smoking and/or between marihuana use and cigarette smoking in adolescents and college students have also been identified in a number of other investigations (86,97,153, 177,181).

DEMOGRAPHIC AND PSYCHOSOCIAL CORRELATES OF SMOKING IN ADOLESCENCE

Smoking is a complex behavior, and it is likely that adolescents start to smoke for multiple reasons. Strong correlations between smoking and a number of demographic and psychosocial variables have been reported, but causal connections have not been established. Neither has the set of "predisposing factors" been often subjected to multivariate analysis. It is rare that more than one or two variables have been tested simultaneously. What appear to be separate determinants of smoking behavior (for example, peer pressure and socioeconomic status) may actually be reflecting a single underlying pattern. For example, aspects of self-confidence, academic achievement, types of parental and/or peer relations, and/or socioeconomic factors cluster in certain ways to influence susceptibility to smoking cigarettes. A few multivariate analyses have been conducted (111,113,138).

Socioeconomic Influences

A number of studies have examined smoking in relation to socioeconomic status. The findings consistently point to a relationship between lower parental status—income and education—and higher smoking prevalence among these parents and their children (20,130,148,161). Adolescents from low-income families may also begin to smoke earlier than others (33,126). The findings that girls who work have higher rates of smoking may also reflect a relationship to lower economic status (9,130). Srole and Fischer observed a relationship between downward mobility and smoking in adults (180). This may be an important dynamic to explore in adolescent initiation of smoking.

A relationship between parental education and adolescent smoking also exists (130). When one or both parents attended college, 9.9 percent of boys and 10.6 percent of girls smoked, compared with 10.9 percent of boys and 14.8 percent of girls from homes where neither parent attended college.

Family Patterns

In single-parent households (19.3 percent of those households surveyed in 1979), adolescent smoking rates were approximately double those of households in which both parents were present (130). This relationship holds for both boys and girls, in every age group, and across all five NCSH/NIE surveys; it has also been identified by others (111). In the 1979 survey, 19.3 percent of the boys and 21.2 percent of the girls in single-parent households are smokers, compared to 8.6 percent and 10.7 percent of those in homes with both parents present.

Parental modeling may underlie this association in two ways. First, adult smoking rates are higher for divorced or separated men and women. Second, female single parents who head households are likely to work outside the home, and smoking is more prevalent among working women than among homemakers (182).

Smoking Among Parents and Siblings

Adolescents are more likely to smoke if either or both parents smoke than if they do not (9,15,20,161,213). In the 1979 NIE Survey this pattern was found across age and gender (130) (See Table 3.) Looking at the data slightly differently, when both parents smoke, 13.5 percent of sons and 15.1 percent of daughters smoke; when one parent smokes, 9.1 percent of boys and 12.7 percent of girls smoke; and in homes where neither parent smokes, 5.6 percent of boys and 6.5 percent of girls smoke (130).

There are conflicting reports on the relationship between the sex of the smoking parent and smoking habits of the offspring. In two-parent homes in which only one parent smokes, 17 to 18 years olds appear to be more likely to smoke if the mother does (130). Other studies have identified a relationship between the child's smoking and that of the parent of the same sex (9,15,213). Allegrante, et al. found a relationship between the mother's smoking behavior and that of sons, but not of daughters, and no relationship of the father's smoking behavior to smoking by children of either sex (3). In contrast to all of these findings, Schneider, et al. were unable to relate parental smoking to that of offspring (166).

Explanations for the association between parental and children's smoking behavior include the effect of role-modeling, pa-

TABLE 3.—Percentage of adolescents who smoke by the smoking behavior of parents and older siblings

	Have No Older Sibling One or Both Parents Smoke	Have No Older Sibling Neither Parent Smokes	Older Sibling Smokes One or Both Parents Smoke	Older Sibling Does Not Smoke One or Both Parents Smoke	Older Sibling Smokes Neither Parent Smokes	Older Sibling Does Not Smoke Neither Parent Smokes
Boys:						
12–14	2.8	0.0	6.3	2.7	0.0	0.0
15–16	17.6	4.0	18.8	6.3	21.1	2.1
17–18	15.0	7.9	25.4	16.7	31.7	0.0
Total	8.2	2.9	17.0	7.5	19.5	0.6
Girls:						
12–14	3.7	0.0	8.5	1.3	3.4	2.9
15–16	8.2	5.7	20.0	13.0	15.2	2.4
17–18	29.7	15.4	32.9	19.6	25.0	6.7
Total	9.7	4.1	20.3	9.7	15.3	4.1

Base: Both parents present in household
SOURCE: National Institute of Education (130).

rental permissiveness (real or imagined), and availability of cigarettes in the home (125).

Older siblings seem equally important or more important than parents as potential role models for smoking (9,130,148). There is a greater likelihood that an adolescent will smoke if one or more older siblings smoke than if no older siblings smoke; this is true in those households where neither parent smokes as well as in those where one or both parents smoke. In the 1979 survey, boys with older siblings who smoked were more than three times as likely to smoke as boys with nonsmoking older siblings. The increase is about twofold for girls. The highest smoking rate for girls was found when at least one parent and an older sibling smoked (20.3 percent). The corresponding rate for boys (17.0 percent) was slightly lower than where an older sibling but neither parent smoked (19.5 percent) (130). (See Table 3.)

Peer Group Influence

Adolescents' smoking behavior is highly correlated with reports of having friends who also smoke (15,132,133,155,162,216). Most multivariate analyses have established this factor as being of prime importance although one such analysis found no relationship at all (3,113,138). It has been pointed out that patterns of drug use in adolescents are very similar among best friends (121). It has not been demonstrated, however, that it is the behavior of friends rather than inclinations of the adolescent which influences him or her to smoke (3,130,166).

Inquiring about the smoking behavior of the "four best friends" of adolescent respondents, the NIE study reported that 87.6 percent of boys and 94.0 percent of girls who smoked stated that at least one of those friends also smoked. In addition, only 10.2 percent of boys and 5.9 percent of girls who smoked had no regular smokers among their four best friends, and an even smaller fraction (2.2 percent of boys and 0 percent of girls) reported that none of their friends had even experimented. In a parallel vein, it was found that nonsmokers also congregate together. Approximately one-third of the nonsmokers (33.8 percent of boys, 32.9 percent of girls) reported having at least one best friend who smoked, while over two-fifths (43.0 percent of boys, 44.1 percent of girls) had no best friend who smoked regularly. Over one-fifth (22.4 percent of boys, 23.0 percent of girls) had no best friends who had even experimented.

Thus, "peer pressure" to smoke may be operative when the adolescent belongs to or would like to belong to a group in which smoking is part of the life-style (130). When the peer group behavior does not include smoking, there may be little pressure on the adolescent to begin to smoke.

Conformity pressures and peer influence are very strong in early adolescence. Therefore, if smoking were considered a behavior which was adopted by the majority of adolescents, experimentation and initiation might occur because of the importance of conformity in this age period (63). Unfortunately, there are suggestions that most adolescents tend to overestimate the proportion of their peers who are smokers. Eighty-two percent of all girls surveyed in the 1975 American Cancer Society Survey thought of adolescents as smokers rather than nonsmokers (216). In that same survey, the professions of teachers, executives, housewives, and feminist leaders were all characterized as smokers by approximately two-thirds of girls, with only doctors and athletes considered nonsmokers.

Heterosexual peer considerations may also be important. Girl smokers are very likely to have boyfriends who also smoke (72 percent), compared with nonsmoking girls (27 percent) (216). Similar percentages apply to the fraction of all male friends who smoke (69 percent for girl smokers and 32 percent for nonsmokers). Yet girls are less likely than boys to see smoking as a social asset (37 percent versus 55 percent) and they even consider it a drawback (52 percent girls versus 31 percent boys).

The kinds of images projected by the people shown in cigarette advertisements may lend support to peer influences to smoke. Girl smokers characterized such people as attractive (69 percent), enjoying themselves (66 percent), well-dressed (66 percent), sexy (54 percent), young (50 percent), and healthy (49 percent).

Prevention efforts aimed at making actual statistics on smoking prevalence available to teens in order to correct the above beliefs may help counter the advertising. Popular personages in various professions and lifestyles which girls mistakenly perceive as smoker-dominated could be recruited in this effort.

Scholastic Achievement and Aspiration

Achievement in school has been one of the most frequently investigated correlates of smoking, with a study as early as 1923 showing an association between poor school grades and smoking (15,83,121,137,143,161,212). Two studies have reported this association specifically for girls (35,216). Comparing the three factors—parental smoking, socioeconomic status, and scholastic performance—Borland and Rudolph identified scholastic performance as the strongest correlate of smoking in a sample of high school students (20). Studies of achievement, aspirations and expectations in relation to smoking have found that reduced motivation and lower aspiration are associated with a higher prevalence of smoking (3,33,101,130). High school students in college preparatory courses were far less likely to smoke than students in any other type of curriculum (130). Smoking rates for boys and girls preparing for college (9.0 percent and 12.0 percent, respectively) were 50 to 60 percent of those in other curricula (18.3 percent of boys, 20.1 percent of girls). The same trend was found in a previous study (216). Smokers are less involved in extracurricular school activities and have a higher rate of absenteeism (9,35,137).

These factors are undoubtedly interrelated with social class and other factors. Sense of competency and sense of efficacy (or personal control) are linked to school achievement. Smokers have been reported to have less confidence that they can control what they will become (130). McAlister, et al. comment that high academic achievement is probably also associated with admission into a peer group in which smoking is not accepted (125). Furthermore, they state, "Educationally deprived young people may be somewhat less aware of the risks of smoking, but they also experience more stress and greater pressure to adopt behaviors that signal independence and maturity" (125).

Dynamic/Personality Factors

Up to this point, adolescent smoking has been described and analyzed in terms of discrete variables, many of which are truly not independent of one another. From them, a composite picture of the environment of the female smoker begins to emerge. Paralleling the behavioral descriptors is a set of individual/personality factors which include attitudes, values, beliefs, and perceptions which relate the adolescent to the world around her. Vitally important are feelings of self-worth, aspirations and expectations for the future, and feelings of efficacy, competence and the girl's view of her own smoking behavior.

Yankelovich, et al. have provided a thought-provoking description of the evolution in values which has occurred over the past 20 years (216). Smoking is just one behavior which may have been "suppressed" by social norms prescribing appropriate behavior for women in the past, and which now may be "disinhibited" in a very real sense.

Accompanying this shift in sanctions on female behavior is an increase in expressed rebelliousness among girl smokers, which was formerly more characteristic of boys. A higher percentage of female smokers than nonsmokers are annoyed by "experts" who define what is good for them (53 percent versus 34 percent), agree that there is too much regulation of people's lives (50 percent versus 39 percent), and do not want to follow their parent's wishes regarding their behavior (almost 50 percent versus 26 percent) (216). Factor scores of male and female smokers similarly reflect a more negative "feeling toward authority" or dislike of adult-imposed restrictions than those of nonsmokers, and are approximately equal for both sexes (130). Clausen noted that girls who smoked were less acquiescent to their parents, more autonomous, and "strikingly higher in quest for power" than nonsmoking girls (33).

The evolution in values and sex-role behaviors has resulted in some interesting differences between male and female smokers (216). The male smoker remains more socially uneasy, expresses a greater need to be popular with the opposite sex, and considers smoking more of a social asset than the female smoker. The female smoker, compared with her nonsmoking peer, is more likely to consider parties a favorite leisure time activity, to have a boyfriend, and to have had sexual relationships (see also 174). In addition, she is less likely to feel nervous meeting new people. Finally, while she is more willing to admit that smoking is a drawback, she shows less acceptance than the male smoker of the stereotype that adolescents begin to smoke cigarettes to gain peer acceptance and approval (130,216). Nonsmokers show the greatest acceptance of this stereotype and the one which describes the smoker as a "show-off" (216), who believes that smoking makes one look "cool" or "grown-up."

In other studies of smoking behavior, self-esteem has usually been investigated in terms of the adolescent's self-confidence in interpersonal relationships. Smoking is ego enhancing and facilitates social functioning (122,123). This has been observed specifically among adolescent girls and female undergraduates who smoke (174,216). Smoking is correlated with a wish to be older (130). Both boys and girls who differed from the norms of their high school peers on tests of self-concept were more likely to smoke cigarettes as well as to use other drugs (95).

Adolescent smoking has been consistently correlated with low educational and occupational aspirations. In a review which included "locus of control" as a measured variable, Smith concluded that smokers were more externally oriented and felt that they had limited control over what happened to them (176). Pflaum reviewed findings on the positive relationship between smoking and feelings of helplessness and hopelessness (143). Adolescent smokers express less desire and ability than nonsmokers to control future events—for example, to determine what kind of person they will become (130). Girls scored slightly higher than boys on this factor, indicating a greater sense of future control.

Finally, response to stress has been suggested as a basic dynamic in cigarette smoking (122). Feelings of unattractiveness, a sense of incompetency and inefficacy in school achievement and personal relations, limited opportunities for personal growth and for future social and economic roles all contribute to stress in adolescence. Changes in social settings, such as transition from elementary to junior high shool, which occur simultaneously with physical and emotional changes must also be acknowledged. Theoretical formulations of life-change events and their effects on health might also be worth considering in studying the onset of cigarette smoking among girls (47).

Prediction of Future Smoking Behavior

In 1979, a longitudinal study was undertaken by the National Institute of Education involving the re-interview of 46.8 percent (N= 1,194) of the 2,553 adolescents first surveyed in 1974 (130). In 1974, 152 respondents were smokers and 1,042 were nonsmokers. By 1979, 27 percent (N = 41) of the smokers had quit, while 73 percent (N = 111) had continued to smoke. During the same time period, 20.8 percent (N = 217) of the nonsmokers had taken up smoking, while 79.2 percent (n = 825) had not. Thus, the proportion of smokers who had quit was greater than the proportion of nonsmokers who had taken up the habit. However, because the percentage of nonsmokers was much higher than the percentage of smokers, the net effect was an increase in the percentage of the population who were smokers (12.7 percent to 27.5 percent).

With each increase in age group, the proportion of boys who initiated smoking became smaller, so that boys who reached age 17 or 18 as nonsmokers were not likely to start in the next five years. Only 15.4 percent did so, compared with 19.3 percent of 15 to 16 year olds, and 21.6 percent of 12 to 14 year olds. For girls, the pattern is less clear. Fifteen to 16 year old nonsmokers in 1974 showed the greatest proportion of initiators (27.1 percent) by 1979. In the 12 to 14 age group, 22.8 percent took up smoking, and only 14.7 percent in the 17 to 18 age group did so.

Demographic and psychosocial relationships studied in 1974 were reexamined in this group now aged 17 to 23. The influence of older siblings became less powerful than the influence of peers, but educational attainment was still inversely correlated with smoking status.

Those smokers who had quit had a shorter lifetime history of smoking and were lighter smokers than those who were current smokers in 1979. Of the former smokers, 24.7 percent said they had been smoking less than daily just before quitting, and another 34.5 percent smoked 1 to 14 cigarettes per day. Only 7.6 percent of current smokers report less than daily consumption. This suggests that the former smokers may have been less dependent (psychologically or physiologically) upon cigarettes and may have found giving up the habit easier than heavier smokers. In fact, 50 percent of the former smokers succeeded in quitting on their first attempt, while 61.6 percent of current smokers had made one or more unsuccessful attempts to quit.

These young smokers were concerned about health issues. Sixty percent of current smokers had made at least one attempt, and another 20 percent would have been willing to quit if there were an easy way to do so. A greater percentage of young women than men (91.0 percent and 85.2 percent, respectively) expressed a concern about health effects of smoking. The risk associated with oral contraceptive use and smoking and the harmful effects on the fetus of smoking during pregnancy (130) may be responsible for this increased concern. Young women were more likely than young men to say that all cigarettes are equally hazardous (33.7 percent and 25.9 percent, respectively).

Multiple regression analysis was used to identify those adolescents most likely to take up smoking, and discriminant function analyses were used to predict future smoking for each stage—nonsmoker, experimenter, regular smoker, and ex-smoker. The best predictor of future smoking behavior was the adolescent's own perception of his or her future smoking behavior.

The best predictors of future smoking for never-smokers and experimenters were smoking by an older sibling, scores on attitude scales, and age. The chance that a nonsmoker will start smoking become smaller as the nonsmoker grows older. Once regular smoking was initiated, the variables of higher dosage, lower educational aspirations, friends who smoked, and lack of acceptance of the health risks of smoking predicted continued smoking behavior.

In summary, this study revealed that former smokers seemed more similar to experimenters than to regular smokers. Their smoking histories were shorter, and they had a lower dosage and did not have much difficulty quitting. Regular smokers, on the other hand, tried to quit or expressed an interest in doing so, and were bothered by the health hazards associated with smoking. Five years previously, they were able to accurately predict their current smoking status. Smoking was also more likely to be a behavior of their older siblings and peers. And lastly, both educational aspirations and attainments were lower for this group.

PREVENTION OF SMOKING AND CONSIDERATIONS FOR FUTURE RESEARCH

Prevention of the Initiation of Smoking

There are a number of ongoing interventions which attempt to prevent the initiation of smoking (34,58,59,125,198). These studies are directed at elementary, junior high, and high school students, and use an "inoculation" approach to prevention. Exposure to a small amount of information about pressures to smoke is accompanied by practice in coping and assertiveness strategies. The main types of influences in which students are instructed are peer pressures, parental modelling, and media pressures. Peer instructors are often used to maximize influence. Compliance in self-reporting smoking behavior is increased by the use of physiological measures of smoking, for example, salivary nicotine or expired air carbon monoxide, which may or may not be analyzed for the entire subject sample.

Dissemination of information about the health risks of smoking seems to be successful, at least on a superficial level. Ninety-six percent of all adolescents (and 91.6 percent of smokers) "strongly or mildly agreed" that smoking is harmful to health (130). Percentages were similar for boys and girls, and nonsmokers scored higher on all health-related questions than smokers. Almost 90 percent of adolescent smokers (87.9 percent of boys and 89.9 percent of girls) "strongly or mildly agreed" with the statement, "I believe the health information about smoking is true." Fishbein has pointed out, however, the potential importance of the difference between strong and mild agreement with such statements, and the lack of direct personal attribution involved (63). Only 60 to 65 percent of adolescent smokers expressed strong agreement, compared with approximately 80 percent of nonsmokers. Either reduction of cognitive dissonance by denial or actual lack of information may underlie this response pattern. Finally, a surprisingly high percentage of smokers feel (strongly or mildly agree) that it is all right to smoke if "you don't smoke too many." On this item, fewer girls (25.6 percent) were willing to endorse this statement than boys (43.3 percent).

Somewhat lower estimates of the acceptance of health information comes from the 1975 American Cancer Society (ACS) Survey (216). Of all adolescent girls 74 percent agree that smoking is as harmful for women as it is for men; 71 percent agree that smoking is harmful for young people as well as for older people; 56 percent agree that it is not safe to smoke low "tar" cigarettes; and 56 percent agree that smoking is as addictive as

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illegal drugs. Comparable figures are not provided for boys, nor are the data broken down by smoking and nonsmoking categories. This survey further reports that 68 percent of the girls sampled were not warned about smoking by their doctors.

While 60 percent of female smokers began to smoke before the age of 13, only 48 percent attended an antismoking education program in school, and a mere 4 percent attended such a program in the sixth grade when they were approximately 12 years old.

These statistics suggest that smoking education and coping strategies should begin earlier in schools and should begin earliest for high risk groups.

Research Goals

The best evidence suggests that female cigarette smoking rates are declining. This change has occurred in more recent adolescent cohorts—those born after 1962. National surveys are likely to underestimate true rates, whether school, household, or telephone samples are used. Drop-out, absenteeism, lack of telephone accessibility, and belonging to a minority group all contribute to the sampling errors, which include underrepresentation of population subgroups whose rates are substantially higher than the norm. Accurately measuring these subgroups would enable scientists to better target interventions. Young black females appear to be one such group whose smoking rates well exceed the national average (33).

There is good reason to expect the heaviest cigarette use and other "problem behaviors" among those segments of the adolescent population who feel cut off from socioeconomic opportunity and mobility. The review of correlates of adolescent smoking shows that many of the variables that predict cigarette smoking bear a remarkable similarity to ones identified as predictors of marihuana and/or other illicit drug use. It is recommended that greater attention be given to models of behavior and socialization processes.

More prospective longitudinal studies need to be undertaken, based on varied samples of children. Data need to be collected about physical and emotional status, psychosocial outlooks and attitudes, family and peer relations, academic and recreational activities, family and school settings, and family and residential background. This information must be gathered early in childhood to record significant socialization influences which precede the onset of smoking behaviors and should be collected frequently enough to record significant changes close to the time they occur.

TABLE 4.—Smoking parameters observed in Hamburg, Germany, in 1971 and 1974

	Puff Number		Puff Duration (sec)		Puff Interval (sec)		Total Puff Duration (sec)	
	1971	1974	1971	1974	1971	1974	1971	1974
Men	10.2	10.9	1.47	1.47	52.9	42.1	15.0	16.0
Women	10.9	13.3	1.31	1.17	46.0	40.7	14.3	15.5
All	10.5	11.8	1.41	1.34	50.3	41.5	14.8	15.8

SOURCE: Schulz, W. (167).

PATTERNS OF CIGARETTE SMOKING

Smoking patterns differ between the sexes. Schulz and Seehofer studied the smoking behavior of male and female smokers observed surreptitiously in public places. Puff number, duration and interval were measured (167). Women were found to leave a significantly longer butt length (approximately 2 mm longer) and had shorter puff durations than men (Table 4). However, they took a greater number of puffs and, therefore, had the same total puff duration (puff number x puff duration). These authors do not report gender data on inhalation patterns, which are crucial to determining dose. However, Creighton and Lewis reported no sex differences in puff volume in a small study of the inhalation patterns of eight men and eight women (39).

Data on smoking patterns were collected in surveys conducted in 1964, 1966, 1970 and 1975 by the National Clearinghouse for Smoking and Health (NCHS) (see Table 5). In each survey a greater proprotion of men than women reported inhaling deeply into the chest and inhaling almost every puff. Men therefore may extract a greater dose of nicotine and the other constituents of cigarette smoke than do women. However, there is an increasing proportion of women who report smoking their cigarettes "as far as possible," in contrast to a decline in the proportion of men who reported this behavior (167,192,193,194). A slightly higher proportion of males reported letting "very little" of their cigarette burn without smoking it: 1970, 20.6 percent male vs. 18.0 percent female; 1975, 20.9 percent male vs. 18.6 percent female (193,194). These changes are often a correlate of heavier smoking. In sum, the observational data suggest that men and women have equal total duration of smoking per cigarette, and the national survey data suggest a larger proportion of males inhale deeply. In general, men smoke in a more hazardous way than do women. However, the smoking patterns of women are changing toward "more hazardous" smoking (see Part I of this Report).

In contrast to the minor changes that have occurred in the way an individual cigarette is smoked, there have been substantial changes in the percentage of both male and female smokers who smoke more than a pack per day (Table 6). A number of explanations may be offered for these data: (1) more lighter than heavier smokers may be quitting, resulting in a mean increase in daily consumption; (2) continuing smokers may be increasing consumption; (3) smokers newly initiating the behavior may be smoking more heavily than already estab-

TABLE 5.—Respondent-reported styles of cigarette smoking, current, regular cigarette smokers, selected categories, adults, United States, 1964–1975

	1964		1966		1970		1975	
	Male	Female	Male	Female	Male	Female	Male	Female
1. Inhaling deeply into the chest	36.5%	22.5%	31.8%	15.5%	34.3%	17.5%	30.3%	16.4%
2. Inhaling almost every puff	63.1	54.8	63.0	52.1	60.5	47.2	58.5	50.7
3. Smoking cigarette as far as possible	15.9	7.5	13.5	10.0	9.6	10.4	10.9	12.9

1. In 1964 and 1966, the questionnaire response was "as deeply into the chest as possible." In 1970 and 1975, the questionnaire response was phrased "deeply into the chest."

2. In each survey year, the questionnaire response was "inhale almost every puff of each cigarette."

3. In 1964 and 1966, the respondent was asked to draw a line on a diagram of a cigarette, indicating the average length of the discarded cigarette butt length. In 1970 and 1975 the verbal questionnaire response was smoking cigarette "as far as possible." The data for 1964 and 1966 correspond to those respondents indicating a discarded cigarette butt length no greater than 20 mm.

SOURCE: National Clearinghouse for Smoking and Health (192,193,194).

TABLE 6.—Estimates of the percentage of current, regular cigarette smokers who consume more than one pack per day, adults, United States, 1955–1976

Year	Supplement to Current Population Survey (17 yrs. and over) 21 cigarettes or more daily			Health Interview Survey (17 yrs. and over) 25 cigarettes or more daily			National Clearinghouse for Smoking and Health (21 yrs. and over) 25 cigarettes or more daily		
	Total	Male	Female	Total	Male	Female	Total	Male	Female
1955	20.2[1]	25.5	9.8						
1964							25.7	32.4	17.7
1965				19.9	24.5	13.7			
1966	21.6	26.3	15.7				27.2	34.7	16.9
1967	21.9	26.2	16.3						
1968	22.4	26.5	16.8						
1970				23.3	27.6	18.1	25.2	31.1	17.1
1974				24.7[2]	30.3	18.4			
1975							30.1	36.0	22.8
1976				25.3[3]	30.8	19.4			

[1] 18 years and over.

[2] Data provided by Health Interview Survey, National Center for Health Statistics.

[3] 20 years and over.

SOURCE: U.S. Department of Health, Education, and Welfare (198).

TABLE 7.—Estimates of the percentage of current, regular cigarette smokers among white and black adults, aged 20 years and over, United States, 1965–1978

Year	White		Black	
	Male	Female	Male	Female
1965	51.5	34.2	60.8	34.4
1970	43.7	31.9	54.0	33.1
1974	41.9	31.8	55.3	36.8
1976	41.2	31.8	50.5	35.1
1978*	36.4	30.1	42.8	30.2

*NOTE: Results displayed as percentage of respondents with known smoking status aged 17 years and over.
SOURCE: U.S. Department of Health, Education, and Welfare (198).

lished smokers; and (4) declining "tar" and nicotine contents of cigarettes may be leading to compensatory increases in number of cigarettes smoked in order to maintain nicotine dosage (198).

Regarding type of cigarette smoked, the 1975 NCSH survey reported that more women than men smoked filter tip cigarettes (all types), 90.6 percent vs. 79.3 percent. Women seem to be innovators in changing smoking practices. Sixty-one percent of women and only 10 percent of men acknowledge changing brands at least once, and women lead the trend in adopting king-size, filter-tip and 100 mm cigarettes. On the other hand, women smoke cigarettes almost exclusively. Cigars and pipes are currently used by 18 percent and 25 percent of men, respectively, but by less than 0.5 percent of women. Less than 2 percent of women use snuff or chewing tobacco compared with 2.5 percent and 4.9 percent of men, respectively.

SMOKING PREVALENCE AND ETHNICITY

The prevalence of smoking in the population varies not only with age, sex, and socioeconomic status, but also with race and cultural background.

Table 7 presents smoking prevalence among white and black adults form 1965 to 1978 (198). Smoking has declined among men of both races, but prevalence has decreased only slightly among white and black females. Congruent estimates of prevalence and lower cessation rates among blacks have been obtained in other studies (66,183,201).

Despite their greater prevalence of smoking, black men and women smoke fewer cigarettes per day than whites (66,183).

Black women may suffer the worst aspects of sexism and racism with respect to occupational opportunity and financial compensation. Cigarette smoking may be related to assertion, independence, and rebellion or to identification with behavioral patterns of black males. Adolescent dynamics have been studied more than those of adults (see the section on adolescent smoking cessation in this Part). Warnecke, et al. found that social and psychological correlates among black women are similar to those observed among white women (201).

Friedman, et al. examined smoking prevalence among Asian men and women—Chinese, Japanese, Korean or unknown—from the Kaiser Permanente Health Plan and found a smaller percentage of cigarette smokers than among whites or blacks. Asian women had the least frequency of current, established cigarette smokers, 23.1 percent, compared to 39.2 percent of white women and 42.1 percent of black women. Asians were also the least likely to inhale among most age-sex groups of smokers. There were fewer cigarette smokers among Chinese than among Japanese; this was particularly true for women and younger men (66).

PHARMACOLOGICAL EFFECTS OF SMOKING

One or more of the constitutents of cigarette smoke may play a role in the maintenance of smoking behavior and help account for the difficulties many individuals experience when they try to quit smoking (198).

Nicotine

Nicotine is absorbed rapidly from the oral and intestinal mucosa, lungs, and skin. It is distributed throughout the body and is metabolized by several organs, including the liver. It is then rapidly cleared, primarily through the kidney. Nicotine has effects on several organ systems, including the autonomic nervous system, voluntary muscles, stomach, intestines, heart, and brain. Most of the pharmacological actions of nicotine are thought to result from its interaction with receptors of cholinergic nervous systems. Analysis of the physiological effects of nicotine is complicated by the abundance of those effects. Many organs receive input from several neuronal systems which are altered directly or indirectly by cholinergic activity. Furthermore, the effects of nicotine itself depend both on the dose and on the time course of drug administration: brief exposure or low doses cause excitation of cholinergic systems, while long exposure and high doses result in inhibition and paralysis.

Peripheral Effects

Nicotine produces a variety of changes in the autonomic nervous system due to simultaneous effects on both sympathetic and parasympathetic systems. The end result is an increased heart rate and blood pressure; cold, clammy skin; increased acid production in the stomach; increased intestinal activity; and biphasic changes in salivation, with an initial increase followed by a decrease. Nicotine also increases respiration.

Central Effects

Nicotine produces tremors and causes water retention by a central effect on antidiuretic hormone release. Nicotine-induced nausea and vomiting reflect a complex interaction between central and peripheral effects. To date, no specific effects on complex emotions and behaviors have been demonstrated. Animals will self-administer nicotine under certain circumstances, indicating that it may have pleasurable effects.

A Possible Role for Nicotine in Smoking Maintenance

A strong argument has been made for classifying smoking as an addiction, with nicotine as the leading candidate for the addictive agent. Inhalation of cigarette smoke offers an effective way to administer nicotine. Absorbed rapidly, it travels as a highly concentrated bolus through the heart and directly to the brain and is then rapidly cleared. A smoker who smokes one pack per day can average around 70,000 such nicotine "injections" per year. In behavioral terms, smoking has many potential conditioned stimuli, ranging from the taste, sight, and feel of the cigarette itself, to the many social settings in which smoking takes place. If nicotine were a strong unconditioned stimulus, particularly when inhaled, then it would be easily understandable that smoking can become a remarkably persistent habit through connection of this unconditioned stimulus with the many associated stimuli.

Although nicotine has effects on essentially all major organs in the body, including the brain, the role of those actions in maintaining the smoking habit remains an important but unresolved area of research.

The nicotine hypothesis of smoking states that the pharmacological actions of nicotine are "reinforcing." The most likely site of this rewarding or reinforcing action is the brain, with the precise locus of reinforcement not yet determined. Inhaling smoke insures rapid delivery of nicotine to the brain. It takes approximately 13.5 seconds for an intravenous injection of nicotine in the arm to reach the brain; but by inhalation, the delivery time is 7.5 seconds (158). The plasma half-life of nicotine is approximately 30 minutes, and the pack-a-day smoker lights up approximately every 30 to 40 minutes of the day. This suggests that the smoker is attempting to maintain a constant level of nicotine.

The nature of the reinforcing effect is sometimes described as an alteration of arousal. Stimulation may be subjectively experienced as increased alertness, a facilitation of concentration, or an aid to continued efficient performance in fatiguing tasks. Sedation, on the other hand, may be experienced as a tranquilizing or calming effect or as a reduction of some dysphoric state, such as anger. Smoking has been described as distinctly pleasurable following a meal or accompanying xanthines (coffee and tea) or alcohol. Pharmacologic and psychologic components to these subjective reports are beginning to be identified (70,78).

There is extensive literature describing acute and chronic nicotine administration in animals including a limited number of self-administration models. Tolerance to nicotine has also been described (81,88,112).

A number of studies have examined the hypothesis that humans self-administer tobacco in order to obtain nicotine. Studies have also examined compensatory adjustments in the number of cigarettes and manner of smoking by subjects in response to experimenter-induced increases or decreases in cigarette nicotine content, cigarette size, availability, or supplemental nicotine administration. Chewing gum containing nicotine, nicotine tablets, intravenous nicotine and central or peripheral nicotinic blocking agents have been used to supplement or block the effects of the nicotine absorbed from the smoke. A titration effect is said to occur if subjects change their cigarette smoke intake in the appropriate direction in response to these experimental manipulations.

A modest amount of compensation has usually been demonstrated (79,158). Smokers seem to titrate along the nicotine, rather than the "tar" continuum but an optimum ratio of nicotine to "tar" probably exists for effective delivery to the lung. Experiments involving the intravenous administration of nicotine have been inconclusive, with both positive and negative effects on the suppression of subsequent smoking having been observed. When compensation occurs, it is seldom complete. This may be due to a number of factors: (1) the inability to accurately measure the smoker and/or nicotine dose delivered to the subject; (2) technical problems in experimental design (79,198); (3) secondary reinforcing effects of smoking which mask titration; and (4) the fact that people may smoke for reasons other than regulation of nicotine level.

Some have even suggested that nicotine controls smoking behavior only at the extremes, and then as an aversive agent (163).

Too much smoking might lead to such high serum concentrations of nicotine that toxic effects encourage lower intake; and too little smoking or smoking of low-nicotine cigarettes could lead to such low concentrations that withdrawal side effects encourage resumption of smoking. This hypothesis states that, between those two extremes, other factors such as psychological and social pressures are far more influential in determining smoking patterns.

Differences in Nicotine Metabolism

The metabolism of nicotine may be different in men and women. Measurement of nicotine and cotinine (the principal metabolite of nicotine) excreted in the urine after intravenous administration of nicotine hydrogen tartrate suggested differences in metabolism based on sex and smoking status (73). In nonsmokers, men excreted less nicotine but more cotinine than women, suggesting greater initial metabolism among men. However, there were no clear differences between male and female smokers.

Schievelbein, et al. studied nicotine and cotinine excretion in both regular smokers and nonsmokers after they smoked cigarettes with differing tar and nicotine levels (165). Women excreted significantly lower amounts of nicotine and cotinine compared with men for three of the four brands tested. The gender difference was found for the excretion of nicotine and cotinine when tested separately and together. The number of cigarettes smoked per day did not differ between the sexes, but the carboxyhemoglobin (COHb) levels, which are often taken as a correlate of depth of inhalation, were lower in the women. The female subjects, therefore, may have received a lower dose of nicotine because of a different smoking pattern.

SMOKING AND STIMULATION EFFECTS

The literature suggests that women are more likely to smoke in situations of high arousal than low arousal and when experiencing "negative affect" (69,96). The effects of smoking, which are often perceived as tranquilizing, might then be sought as a major coping mechanism. However, it can also be argued that the stimulant effects of nicotine, which are usually considered the predominant central nervous system action, might be equally useful as a mobilizer. These related and commonly held beliefs will be examined in some depth.

Frith (69) studied British male and female employees in a psychiatric institute; they ranged in age from 28 to 50. Subjects rated the strength of the desire to smoke in 22 hypothetical situations. The 12 high-arousal items involved either emotional strain and anxiety or demanding mental activity; the ten low-arousal items concerned boredom and relaxation or repetitive tasks and physical fatigue. A factor analysis of the entire questionnaire and t-tests performed on male versus female scores for the most extreme situations on the continuum led Frith to state that men had a greater desire to smoke in situations inducing boredom and tiredness and women had a greater desire to smoke in stress-inducing situations. However, men rated the desire to smoke significantly higher than did women on all three of the questions representing low-arousal situations, whereas women rated the desire to smoke significantly higher on only one of the three questions representing the high-arousal extreme of the continuum (69).

Using Frith's questionnaire, Barnes and Fishlinsky were unable to replicate his findings in a sample of Canadian undergraduates (12). Within the male sample, there was no significant relationship between desire to smoke and the arousal value of the situation in the question, and female subjects indicated a greater desire to smoke in the low-arousal situations. The authors point out the possible importance of sampling differences.

Elgerot studied light, medium, and heavy smokers in an attempt to control potential differences in inhalation patterns between men and women (cited by Frith as a possible explanation for his results) (57). Subjects were Swedish university students. The 42-item questionnaire was similar, but not identical, to Frith's. There was no gender difference for low-arousal situations. There was no sex difference in the light and medium smoker subgroups, but women in the heavy smoker subgroup expressed a greater desire to smoke in stress-inducing circumstances.

Russell and his colleagues devised a 34-item questionnaire covering a wide variety of smoking motives. It was administered to 175 normal smokers and then subjected to factor analysis (160). Six factors, representing six types of smoking, were identified. Women scored significantly lower on what was termed "sensorimotor" smoking, and significantly higher on "sedative" smoking. Thus, the sex difference on "sedative" smoking (reduction of arousal) was supported.

Ikard and Tomkins (96) found evidence that women smoke in situations involving negative affect. Negative affect smoking is defined as smoking which serves to reduce unpleasant feelings. It includes smoking to reduce the dysphoric feelings accompanying rejection by a social group as well as smoking to satisfy a craving for a cigarette (i.e., deprivation negative affect). Positive affect smoking involves the arousal of pleasant feelings.

For example, smoking from curiosity would be classified this way because of the feelings of excitement and interest generated. Ikard and Tomkins showed two films, one intended to evoke positive affect (a slapstick comedy), and another to evoke negative affect (a documentary on Nazi atrocities) to college students who smoke. To be characterized as either positive- or negative-affect smokers, the subjects had to smoke during the appropriate film and indicate a congruent mood on an affect checklist. The major finding was that 73 percent of the female sample of 15 subjects exhibited solely negative-affect smoking compared to only 36 percent of the sample of 39 males. While 80 percent of the females indicated that they were likely to smoke in positive as well as negative-affect conditions, their behavior did not match the self-report in this experiment. It is difficult to determine if the environment of the experiment altered normal behavior patterns, or if perhaps smokers are not accurate in describing the types of situations in which they smoke.

Nationwide surveys conducted in 1964, 1966, and 1970 also suggested that a higher percentage of women than men are negative-affect smokers and that little or no difference exists between men and women in the percentage who are positive-affect smokers (192,193). A greater percentage of women current smokers endorsed the statement, "It relaxes me." (192). This supports the hypothesis that reduction of negative affect is a more important factor for women smokers. The statements assessing positive-affect smoking did not show a clear gender difference. In 1964, slightly more men than women endorsed the statement "enjoys it" as a reason for smoking, but in 1966 there was no difference between sexes and in 1970 slightly more female than male current smokers agreed that "cigarettes are pleasurable" (79.6 percent of women versus 77.0 percent of men).

To summarize: smoking affects arousal; it is not known whether women smoke to maintain a given arousal level, to change that level, or to adjust a physical blood level of nicotine. There are a number of studies which suggest that women use cigarettes more in high-arousal situations than do men. Studies which combine self-report with experimental situations providing a good approximation of natural smoking conditions are needed to shed some light on the validity of evaluation by questionnaire alone.

Smoking Cessation

There is an assumption in the treatment literature that men have greater success than women in quitting smoking. The basis of this assertion lies partially in the demographic analyses of cessation rates and partially in the literature on smoking cessation clinics and experimental programs.

This section presents the results of both demographic and experimental analyses of smoking cessation. A critical appraisal is made of the relative success of men and women in giving up smoking and in remaining ex-smokers. Psychosocial and behavioral factors relating to abstinence and difficulties encountered in quitting are discussed. Finally, recommendations are presented for treatment and future research.

DEMOGRAPHICS

The quitting rates of smokers are calculated by dividing the number of former smokers by the number of ever smokers within each relevant demographic category. The following statistics are taken from the 1975 U.S. Department of Health, Education, and Welfare (USDHEW) survey on Adult Use of Tobacco (194). Former smokers are defined as those who once smoked but no longer do so. The term "former smokers" includes both those who have quit on their own and those who have received outside help. Quitting rates of women lag behind those of men, for each category reviewed.

Age

The USDHEW tables divide adult age groups into six categories: ages 21 to 24, 25 to 34, 35 to 44, 45 to 54, 55 to 64, and 65 and over (194). There is a trend toward increasingly larger percentages of former smokers in each successive age group for both men and women. However, within each age group, the percentage of smokers who have quit is higher for men than it is for women. For example, in the youngest age category, the percentage of female smokers who have quit is 22.6 percent while that for males is 27.9 percent. For a middle-aged category (45 to 54), the female and male percentages are 32.0 percent and 46.7 percent respectively. In the oldest age group, 51 percent of female ever smokers are former smokers, whereas the percentage is 60 percent for males. Bosse and Rose state that the sex differences in quitting are vanishing at younger ages, but Dicken argues persuasively that the absolute amount of convergence is small, and that men remain substantially more likely to stop smoking than women (21,45).

Education

Higher levels of education are associated with higher rates of quitting for both men and women. Among those with a college

TABLE 8.—Most frequently endorsed reasons for resuming smoking· Fall 1964 and Spring 1966 household interview survey, responses of current smokers

Q: People give all sorts of reasons for either not being able to or not wanting to stay off cigarettes. What were your reasons for going back to cigarettes? (Asked if made a serious attempt to stop smoking.)

		Current Smokers			
		1964		1966	
		N	%	N	%
Selected total	M	705	55.7	772	54.9
	F	542	50.6	588	57.1
No will power	M	291	23.0	278	19.8
	F	209	19.5	191	18.5
It relaxes me	M	212	16.8	181	12.9
	F	245	22.9	192	18.6
Enjoys it	M	144	11.4	123	8.7
	F	102	9.5	90	8.7
Helps keep weight down	M	65	5.1	40	2.8
	F	75	7.0	57	5.5
Smoke to be sociable	M	98	7.7	43	3.1
	F	70	6.5	46	4.5

NOTE: More than one answer was allowable for each respondent.
SOURCE: U.S. Department of Health, Education, and Welfare (192).

education or higher, 52.1 percent of the men and 48.1 percent of the women who have ever smoked have quit. For all other levels of education, 40.5 percent of men smokers and 31.3 percent of women smokers have given up smoking. Although the discrepancy is less in the most advanced education category, the percentage of female quitters is smaller at both levels of schooling.

Income

Higher levels of income are associated in both sexes with higher rates of cessation. For those ever smokers with incomes under $10,000, the rates of quitting for men and women are 34.7 percent and 30.3 percent respectively. For those with incomes of $10,000 or above, the rates are 45.7 percent for men and 36.2 percent for women. Quitting rates of men exceed those of women for all but one ($5,000 to $7,499) of the seven income levels.

Occupation

There is a difference of only 7.6 percentage points between the proportion of male and female quitters in the category of professional, technical, and kindred workers, with the male quitting rate at 49.4 percent and the female quitting rate at 41.8 percent. A dramatic increase in this difference occurs, however, among managers, officials, and proprietors. In this category the quitting rate for men is 47.1 percent and that for women is only 26.5 percent. Among sales and clerical workers, 40.1 percent of the men and 25.8 percent of the women have quit. The quitting rate of homemakers (33.9 percent) is in the mid range of the rates for women in other occupations.

In general, then, women are quitting at lower rates than men across the major demographic categories.

PSYCHOLOGY OF CHANGING SMOKING HABITS

A two-year follow-up of over 500 former smokers identified in the 1964 nationwide survey provides support for the demographic data showing higher proportions of ex-smokers among males than females (56). Men were significantly more likely than women to remain successful abstainers. Men and women made approximately the same number of attempts to quit, and current smokers made more attempts than former smokers (168). Furthermore, successful quitters have usually made at least one abortive attempt to quit before succeeding. A survey of young women, aged 18 to 35, revealed that light smokers had the greatest success in stopping smoking (216). This finding is not entirely consistent with that of Eisinger (56), however, who reported that long-term smoking was a predictor of successful abstinence. The difference in study samples may account for the lack of "fit" of the two results, as Eisinger's survey included all adults 21 years of age and older. The "reinterview" (follow-up) aspect of Eisinger's study gives further credence to his conclusions since they are based on data actually obtained at two points in time.

Those factors which consistently seem to differentiate between those who can quit or reduce intake and those who cannot are: the presence of strong motivation and commitment to change; the use of behavioral techniques; and the availability of social support. Those who successfully quit or reduce smoking use behavioral techniques such as substituting candy and gum for cigarettes, and some form of self-reinforcement of desirable behaviors to maintain abstinence (140,216). Successful reducers use behavioral techniques more consistently and for a longer period of time than those who fail to reduce smoking (140). Successful quitters experience cravings when they stop, but the use of substitutes seems partially to alleviate these feelings (139). Furthermore, those smokers who do reduce intake are more motivated and committed to personal change (140), and long-term abstainers have more confidence in their ability to remain ex-smokers (56). Successful reducers receive more positive reinforcement from others and the best known acquaintances of successful abstainers are former smokers (56,140). Warnecke, et al. reported female relatives to be the primary role models for women who quit smoking (201).

TREATMENT STUDIES

Most smokers who attempt to quit do not seek outside help to stop smoking. The population that seeks treatment may be one that experiences severe difficulty in giving up smoking. Thirty-nine treatment studies on smoking have reported success rates for males and females, and have used the criterion of total abstinence. Two exceptions were made for programs that reported "success" in terms of 90 to 100 percent reduction.

The studies reviewed here fall into five categories of treatment: education, physician advice, pharmacotherapy, psychotherapy, and behavior modification (Tables 9-13). The categorization is, by necessity, only a rough separation of treatment modalities. Evaluation of the gender difference question, however, does not rest directly on the categorization schema.

Many of the studies listed in the tables did not report significant evaluations for male/female quitting rates. Therefore, a chi square statistic or Fisher exact probability test was calculated wherever sufficient data were available. Because of the limited number of studies identified for analysis and the often limited sample size, results of borderline ($0.05 < p < 0.10$) and acceptable ($p < 0.05$) levels of significance are reported for the reader's information.

The end-of-treatment cessation rates are high for all types of treatment, but the maintenance of cessation tends to be much lower. In 1971, Hunt, et al. demonstrated that recidivism curves of heroin, alcohol, and smoking are almost identical, with long-term cessation falling off steeply from the end of treatment (94). Within three months approximately 35 percent of successful quitters are still not smoking, and by one year, the figure is closer to 20 percent. In 1978, another reviewer cited virtually the same figures (147). There have been reports of improvement in techniques for obtaining abstinence and in maintaining it, using rapid smoking (an aversive conditioning technique), hypnosis, and group therapy. The long-term cure rates of 60 percent or higher at six months claimed in some studies have not been reproducible in other settings. The smoking cessation literature has been recently reviewed in detail (80,147,168,198).

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Across all treatments, women have more difficulty giving up smoking than men, both at the end of treatment and at long-term points of measurement. No studies have been reported in which women do significantly better than men. Several of the larger studies show higher abstinence rates by men, but many show no difference. Results in the tables are based primarily on those who complete treatment programs. Attrition rates are very difficult to evaluate because most studies do not discuss the issue of subjects who drop out of treatment.

Because of the emphasis placed on the role of physician advice in increasing smoking education and promoting cessation, an estimate of its effectiveness is relevant. From retrospective data, it is estimated that 35 percent of people who have been advised by a doctor either to quit or to cut down sharply, actually do quit (139). Twenty-five percent of those who have not talked to a physician about smoking quit, and only 12 percent who have been told by a physician that it was permissible to continue smoking quit.

The prospective treatment literature yields varying estimates of the impact of physician advice. Ten to 25 percent of patients advised by a physician to quit or cut down actually do so (198). Gender does not seem to exert a particular influence. The primary variables associated with the ability to quit after physician admonition were good psychosocial assets, psychological stability, and the ability to verbalize depression (54).

Success in treatment in general seems to relate to personal characteristics. A shorter smoking history and lower cigarette consumption also predict a greater likelihood of cessation (104,144,204). In addition, those subjects most likely to succeed in treatment are highly motivated, believe they will succeed, and are confident of their ability to stop smoking (82,136,187).

One group of women that seems to have great difficulty in giving up smoking in treatment is homemakers. Homemakers in the age range of 18 to 35 tend to be heavy smokers, and heavy smoking is one predictor of failure in treatment (216). Kanzler, et al. found that homemakers were less successful at quitting, particularly at long-term follow-up (104). However, as previously discussed, homemakers have quit rates in the mid-range of those of women in other occupations; therefore, the difference may apply only to those homemakers who seek help through treatment programs.

Wilhelmsen found significant male/female differences in treatment success rates and stated that the poorer performance of women related almost exclusively to the unsuccessful results of homemakers (209). These women explained that cigarettes served as companions and they reported the difficulties of being

TABLE 9.—Education-Smoking cessation treatment results by sex

			Percent Abstinence		
Study	Treatment	N	End-of-Treatment (%)	Six Months (%)	Long Term (%)
1. Guilford, 1967** (82)	Five-Day Plan* unaided	75M 100F		23M } 2 12F	
	Aided	82M 91F		27M] 1 29F	
2. Peterson et al., 1968** (141)	Five-Day Plan	134M&F	79M&F		19M (18 mo. follow-up 19F on 121 Ss)
3. Berglund, 1969** (4)	Five-Day Plan	895M&F	87M 84F	32M } 2 27F	31M } 1 (4–18 mo.) 23F
4. Delarue, 1973 (44)	Education, small groups	472M&F			34M (12 mo.) 21F
5. Danaher et al., 1978† (41)	Education; skill training group	11F	50 (of 8 Ss finishing treatment)		50 (9 mo.)
6. Ochsner & Damrau, 1970 (136)	Pamphlets*	20M 33F	85M } 1 52F		
7. Pyszka et al., 1973** (146)	American Cancer Society Clinics	131M 223F	39M&F		28M (18 mo.) 20F

TABLE 9.—Education—Smoking cessation treatment results by sex—(Continued)

Study	Treatment	N	Percent Abstinence		
			End-of-Treatment (%)	Six Months (%)	Long Term
8. Kanzler et al., 1976 (104)	Smokenders	210M 343F	70M 69F		57M 30F [1] (48 mo.)
9. Dubren, 1977* (53)	T.V. spots	92M 218F	15M 7F [1]		

[1] $p < 0.05$

[2] $0.05 < p < 0.10$

*Success = 90-100% reduction in smoking.

**Results based only on those completing treatment or contacted for follow-up.

†Pregnancy intervention study.

TABLE 10.—Physician advice—Smoking cessation treatment results by sex

			Percent Abstinence		
Study	Treatment	N	End-of-Treatment (%)	Six Months (%)	Long Term (%)
1. Burns, 1969 (27)	M.D. advice to resp. dis. pts.	66M		53M } 1 (3 mo.)	
		28F		32F	
2. Handel, 1973 (87)	Anti-smoking message in med. exam	45M			38M } 1 (12 mo.)
		55F			11F
3. Burnum, 1974 (28)	M.D. advice	84M	29M		
		40F	18F		
4. Baric et al., 1976 (112)	M.D. advice	134F			
	(spont. quitters)	24	83		
	(intervention)	63	14		
	(control)	47	14		
5. Donovan, 1977†(49)	M.D. advice	552F	50% reduction		

[1] p <0.05

†Pregnancy intervention studies.

without adult company all day and of being deprived of outside activities as obstacles to giving up smoking. Cigarettes have also been described as a means of temporally partitioning the day, of achieving physical autonomy from children, and of providing role differentiation (74).

Frieze, et al. reported women face more life stress than men and have more symptoms of psychological distress (68). Waters reports that women show more overt signs of neuroticism than men (203). Furthermore, he finds an association in women between degree of neuroticism and amount smoked. Burns also found that female smokers had higher neuroticism scores than did female nonsmokers. No such differences were found in men (27).

Some studies have shown that women who smoke are both more subject to psychological stress and more outgoing than women who do not smoke. In a prospective study on women and smoking, Cherry and Kiernan measured personality traits in young women before the onset of smoking (31). They found that smokers had high neuroticism and extroversion scores before taking up the habit. They add that current women smokers are more extroverted and also more neurotic than nonsmokers. There is evidence that women smokers are more independent-minded, assertive, self-opinionated and forthright (151,216). The latter authors report that women smokers are also characterized by apprehension and tension, and that these characteristics are related to an inability to give up smoking.

The presence of psychological distress has also been shown to affect the success of women in treatment. Peterson, et al. found that, while 23 percent of the men who had participated in a smoking program cited nervousness as the principal reason for resuming smoking, 43 percent of the women cited this reason (141). Russell reports that the presence of depression was related to dropping out of treatment, and that depression was more frequent and severe among the women in his sample (156). In a later study, Russell found that within the treatment group, women had worse psychiatric adjustment scores than did men (159). Furthermore, although the degree of psychiatric adjustment did not differ between male treatment successes or failures, treatment successes among women were significantly more likely to have good adjustment scores. Rode found that success in a smoking withdrawal program was related to lack of tension and apprehension for women (150). That smoking might indeed act as a method of coping with psychological and social stress is illustrated by the fear reported by many women that they will engage in symptom substitution—specifically overeating—if they stop smoking (14,23,27). It is also possible that underlying stress in women impedes the strength of the

TABLE 11.—Pharmacotherapy—Smoking cessation treatment results by sex

			Percent Abstinence					
Study	Treatment	N	End-of-Treatment (%)	Six Months (%)	Long Term (%)			
1. Turle, 1958* (191)	Hydroxyzine	23F	4F					
2. Whitehead and Davies, 1964 (208)	Methylphenidate Diazepam	10M 6F	20M 0F		0M 0F	(12 mo.)		
3. Wilhelmsen, 1968 (209)	Methylscopolamine tranquilizer	291M 200F		56M 41F } 1 (12 mo.)				
4. Wetterqvist, 1971* (207) 1973* (206)	Methylscopolamine	192M 98F	50M 33F } 1		19M 12F	(12 mo.)	9M 9F	(60 mo.)
5. Arvidsson, 1971* (5)	Anticholinergics, Group aversion therapy	50M 50F	85M 85F		48M 22F } 1 (12 mo.)			
6. Merry and Preston, 1963* (127)	Lobeline	45M 31F	29M 32F					
7. Golledge, 1965* (72)	Lobeline & placebo	19M 8F	63M 73F					
8. Ross, 1967* (152)	Lobeline Amphetamine	728M 745F	40M 29F } 1		21M 12F } 1 (10–57 wks.)			

TABLE 11—Pharmacotherapy—Smoking cessation treatment results by sex—Continued

Study	Treatment	N	Percent Abstinence End-of-Treatment (%)	 Six Months (%)	 Long Term (%)
9. Schauble et al., 1967* (164)	Lobeline	33M	18M		
	Amphetamine	35F	26F [1]		
	Lobeline, amphetamine	14M	57M		
	and education	17F	26F		
10. West et al., 1977* (204)	Lobeline, amphetamine	255M	43M [1]		22.0M [1] (60 mo.)
		288F	33F		13.4F

[1] p <0.05

*Results based only on those completing treatment or contacted for follow-up.

TABLE 12.—Psychotherapy—Smoking cessation treatment results by sex

Study	Treatment	N	Percent Abstinence: End-of-Treatment (%)	Percent Abstinence: Six Months (%)	Percent Abstinence: Long Term (%)
1. Moses, 1964 (129)	Hypnosis, discussion	35M 15F	83M 53F } [2]	11M 12F	8M 12F (12 mo.)
2. Mann and Janis, 1968 (119)	Emotional role-playing	26F			23–50F (18 mo.)*
3. Streltzer and Koch, 1968 (185)	Emotional role-playing	30F	0F (4 wks. post)		
4. Lichtenstein et al., 1969 (115)	Emotional role-playing	54F	9F (1–5 wks. post)		
5. Fee and Benson, 1971 (62)	Group therapy	306M 204F	56M 38F } [1]		16M 9F } [1] (6–12 mo.)
6. Bozzetti, 1972 (23)	Group therapy	7M 7F	57M 43F		85M 57F (12 mo.)
7. Tamerin, 1972 (187)	Group therapy	16F	69F		

[1] $p < 0.05$

[2] $0.05 < p < 0.1$

*% reduction in smoking.

determination required to cease such behaviors as smoking and overeating. Weight gain is a frequently reported consequence of giving up smoking (173).

THE SMOKING WITHDRAWAL SYNDROME

Few of the studies reviewed here mentioned gender as a connection with withdrawal symptoms, and none suggested that men and women differ in the severity of smoking withdrawal symptoms. However, Shiffman (173) analyzed Guilford's raw data (82), and stated that 15 of the 18 major symptoms reported by subjects demonstrate sex differences (80,173). Thirteen of those 15 symptoms were more frequently reported by women. Other studies show similar, although not statistically significant, trends. (141,190,215).

Factors contributing to relapse, such as craving and nervousness, were reported to be similar for men and women (141). Women who experienced the greatest craving during the initial five days of abstinence were most likely to relapse (82). Since women score higher than men on measures of anxiety as a general rule, it is possible that they would be more susceptible to relapse if smoking had been their customary means of reducing such dysphoria. Women may also pay more attention to somatic symptoms than men, as they make more frequent use of all health care services, and specifically (because of the relative symptomatology) for headache and and weight gain (114).

It is likely that the abstinence syndrome is a major factor in recidivism during the first few weeks of cessation when relapse is most common, and that the number of cigarettes smoked per day is an important variable in determining the severity of the withdrawal. The issue of a gender difference in withdrawal severity is a major area where research is needed.

SMOKING AND WEIGHT CONTROL

Women who smoke are, on the average, thinner than women who do not smoke. The reported mean weight difference ranges from 1.2 to 4.5 pounds (7,17,93). Weight gain has been a frequently documented consequence of quitting smoking, both in males and females, (17,37,65,71,141,190,209,215).

Studies of males have reported weight gains among former smokers which range from 1 to 12 pounds greater than those who continue to smoke. In one such study, the authors observed that, while 60 percent of continuing smokers gained weight, among quitting smokers the observed proportion was 85 percent (37). These figures gave rise to an observed-to-expected ratio of 1.4, suggesting that those who quit are 40 percent more likely to

TABLE 13.—Behavior modification—Smoking cessation treatment results by sex

Study	Treatment	N	Percent Abstinence: End-of-Treatment (%)	Percent Abstinence: Six Months (%)	Percent Abstinence: Long Term
1. Keutzer, 1968 (105)	Breath holding, coverant control, negative practice, attention placebo	73M 73F	18M 29F		
2. Suedfeld and Ikard, 1973 (186)	Sensory deprivation	3M 2F	100M 50F	67M 50F (3 mo.)	
3. Delahunt and Curran, 1976 (43)	Negative practice or self-control	50F	61	22	
	Negative practice and self-control		89	56 ⎫ 1	
	Control		15	0 ⎭	
	Nonspecific treatment		56	11	
4. Tongas et al., 1976* (189)	Covert sensit., smoke aversion, group therapy, combined treatment	38M 34F		71M 39F } 2	62M 32F } 2 (12 mo.) 48M 18F } 2 (24 mo.)
5. Russell, 1970 (156)	Electric shock aversion	10M 4F	70M 50F		40M 50F (12 mo.)

TABLE 13.—Behavior modification—Smoking cessation treatment results by sex—(Continued)

			Percent Abstinence		
Study	Treatment	N	End-of-Treatment (%)	Six Months (%)	Long Term
6. Chapman et al., 1971 (30)	Electric shock, self-management; post-treatment therapist monitoring: 2 weeks:	4M 8F	75M 100F	25M 37F	25M 29F (12 mo.)
	11 weeks:	4M 7F	100M 86F	50M 57F	50M 57F
7. Berecz, 1972 (13)	Electric shock aversion, imagined vs. real smoking	56M 32F	**		
8. Russell et al., 1976 (159)	Electric shock and controls	28M 28F	64M† 57F†		

[1] $p < 0.05$

[2] $0.05 < p < 0.10$

*Results based only on those completing treatment.

**Percent reduction, little for F; more for M in imagined-smoking condition.

†Two weeks post-treatment.

gain weight than those who continue to smoke; but a significant proportion of observed weight gain among men who quit smoking would have occurred even if they had continued to smoke.

The single major report on lifetime smoking and weight patterns in women examined data provided by approximately 57,000 female members of a national weight-reduction program (17). Cross-sectional analysis indicated that current smokers weighed less than nonsmokers by 1.2 pounds and 4.0 pounds less than former smokers. Inhalers were significantly less obese by 5.7 pounds than current smokers who did not inhale. A 40-year longitudinal analysis of weight in relation to reported lifetime smoking history revealed that between ages 30 and 50 (the two decades after the majority of those who quit had discontinued smoking), the former smokers gained more weight than continuing smokers, both for inhalers and non-inhalers. The calculated weight gain after cessation varied substantially by amount smoked; heavy smokers who inhaled (> 41 cigarettes) gained 30 lbs., while light smokers who inhaled (1 to 10 cigarettes) gained only 4 pounds. The observed differences in weight persisted through age 60. Conclusions of this study may not, in fact, be directly applicable to the total female population. This study raises the issues of reporting and recall bias among this obese population (mean group weights ranging from approximately 171 to 180 pounds), as well as self-selection into continuing or former smokers.

The implications of such studies are important. The image of the slender, attractive female pervades our culture and is certainly present in tobacco advertising (84). Do women perceive weight gain as a significant and unavoidable sequel to discontinuing smoking? There is evidence suggesting that fear of weight gain may keep women from quitting smoking. Women are more concerned with weight than men are. In the 1975 NCSH survey, the percentages of female and male smokers who responded "strongly agree" or "mildly agree" to the statement, "Being afraid of gaining a lot of weight keeps people from quitting cigarettes" are shown in Table 14.

Attempts have been made to examine the cause of such reported weight gains. The mechanism of weight gain with cessation of smoking has not, however, been elucidated. Trahair and others have reported that appetite increased with smoking cessation, and the resulting increased caloric intake caused weight gain (190). Other studies have suggested that smoking may, in fact, directly affect metabolism. Glauser, et al. studied seven males before and one month after cessation. Body weight and surface area increased, while heart rate, serum calcium, sugar, and oxygen consumption decreased (71). Conversely, however,

TABLE 14.—Percent affirmative responses to statement: "Being afraid of gaining a lot of weight keeps people from quitting cigarettes"

Smoking Status	Women (%)	Men (%)
Never Smoked	59.0	51.5
Formerly Smoked	63.1	53.6
Currently Smoked	59.9	47.3

SOURCE: National Clearinghouse for Smoking and Health (194).

Sims observed no change in resting metabolic rate, thermic response to exercise or meals, and no change in serum T_3 or T_4 (175).

Further research is necessary to define the degree of weight gain after cessation of smoking, the mechanisms by which it occurs and the ability to modify it by educational or behavioral interventions during and after cessation attempts.

TREATMENT RECOMMENDATIONS

Perri, et al. recommend that smoking cessation programs with a behavioral emphasis be comprehensive, multifaceted, long-term, and that they include self-reinforcement and problem-solving procedures (140). Given the difficulty for some women in simultaneously dieting and attempting to quit smoking, smoking withdrawal programs should adopt a total approach to health, including advice on dieting, exercise and the immediate benefits of abstinence (150).

Marlatt and Gordon write that relapse potential is greater for individuals whose daily schedule fails to include some rewarding or pleasurable activity (120). It would appear useful to attend to this issue in smoking treatment programs.

A social support hypothesis is frequently cited in the treatment literature to explain gender differences in quitting. It is often suggested that women do better than men in programs that provide a maximum amount of social support, and tend to do worse in situations where program support is low or outside factors militate against quitting. For example, Resnikoff, et al. were able to differentiate between those women (but not men) who did poorly in group-plus-medication treatment and those who did well using the Social Introversion Scale of the Minnesota Multiphasic Personality Inventory (149). This scale measures the degree of discomfort in social situations and the presence of outgoing tendencies. Women scoring high on this scale (shyer, more socially introverted) were less likely to quit than low-scoring women. This study provides just one example of the observation that social support seems to be of lesser consequence to men in quitting smoking, although spousal support is important (170).

As the overall categories in Tables 9-13 show, women do more poorly in treatments characterized by less individual attention, such as education and pharmacotherapy, compared with the categories of psychotherapy and behavior modification, where contact is usually maximized in a small group or in an individual-to-therapist setting.

Dubren reports that twice as many women as men participated in a television stop smoking campaign, but that fewer women stopped smoking—presumably because of a lack of support (53). Guilford found that when men and women participated in group programs, success and failure rates were the same for both sexes (78). When they did not attend group programs, men maintained the same success rates, but women achieved markedly lower rates. There is also support for the notion that groups are particularly effective for women if they are sexually homogeneous (44,78). Tamerin writes that the group can provide support, empathy, and shared identification with others going through a similar process (187). The group also provides an avenue for affective expression, so that the relevance of cigarettes to psychosocial events and the personal meaning of giving them up can be discussed. Given the differential reaction of men and women to quitting smoking, as well as the traditionally greater willingness of women to discuss affective issues, it is not surprising that all-female smoking-cessation groups have been particularly attractive.

Marlatt and Gordon studied the circumstances under which smoking relapse is most likely to occur (120). They claim that experiencing stress in the form of a negative emotional state, social pressure, or interpersonal conflict is likely to lead to smoking among those who are attempting to abstain. The occurrence of a full-blown relapse, however, can be attributed to the cognitive reaction to stress-induced smoking. Many individuals who are trying to abstain view a single slip as evidence that they have failed, rather than as a natural and predictable reaction to a stressful situation. Marlatt and Gordon advocate teaching those who are trying to quit the importance of not viewing relapse in an all-or-none manner. Rather, they suggest teaching smokers to "plan for a relapse," to become psychologically prepared to accept a slip as a natural part of the difficult process of quitting.

Another factor that appears to influence the success of women in treatment programs is smoking by significant others in their environment. Kanzler, et al. found a significant trend for women to give up smoking if no one in their daily environment was a regular smoker (104). This trend was only slight for men, although spousal encouragement was related to success in one large study of smoking cessation treatment in men (170). The influence of the smoking behavior of significant others on female attempts to quit has been repeatedly pointed out (14,201,204). Sensitizing friends and relatives who are smokers to this problem, and advising discretion in smoking behavior on their part, might increase treatment effectiveness for women.

CONCLUSIONS

Treatment programs should specifically deal with means of handling anxiety and tension, ways to combat weight gain, and should prepare smokers for mini-relapses. Social support should be maximized. It may be increased through choice of treatment modality, networks of "buddies," friends and relatives, and the involvement of spouses.

It should be possible to capitalize on the heavy commitment of women to the health care system, both in terms of their own use and their role as family providers. Health professionals need to devise targeted interventions for women with this in mind.

Dissemination of Information About Smoking

HEALTH ATTITUDES AND BEHAVIORS

The extraordinarily serious health consequences of smoking have not deterred almost 30 percent of the adult female and 37 percent of the adult male population from smoking regularly. Seventy to 80 percent of these smokers agree that cigarette smoking is harmful, is a health hazard that requires action, and causes disease and death (194). Former smokers and nonsmokers take a much stronger stand on these three points, ranging from 87 to 96 percent agreement. Gender differences are very slight.

The value placed on health compared to other positive life goals was slightly lower for smokers than nonsmokers, and highest for ex-smokers (194). Out of a maximum factor score of six, current smokers averaged 4.66 (M = 4.55, F = 4.81), and nonsmokers averaged 4.82 (M = 4.68, F = 4.9) and ex-smokers averaged 4.89 (M = 4.78, F = 5.06). The higher scores of women support their traditional concern with health in our culture but they are incongruent with recent smoking trends (114).

Fewer current smokers than nonsmokers and ex-smokers report having personally known someone with coronary heart disease, lung cancer or emphysema/chronic bronchitis. This finding may be attributable to a process of denial. Only about one-third of current smokers admitted knowing someone personally whose "health" was adversely affected by smoking while over 60 percent of nonsmokers knew such a person. Clearly, mechanisms must be operating in smokers to reduce cognitive dissonance caused by their behavior and their knowledge of the health consequences of their behavior. One of these mechanisms may be to deny that the health problems of others are connected to smoking.

A related issue is that of compliance. The term encompasses a host of behaviors, all related to following medical recommendations: seeking care when serious symptoms appear, taking medications, having follow-up examinations and procedures, and doing breast self-examination, to name only a few. A large number of studies have been performed in this area, and there is no evidence that one sex shows greater propensity to be compliant than the other (90,114).

Thus, we would have no reason to expect that women and men would respond differentially to doctors' advice to change their smoking behaviors, at least from this literature.

Women in our society are more involved with health care services (114). They arrange for those services and act as role-models for children. This function would have great information delivery potential.

SOURCES OF INFORMATION

There are a variety of ways that people can learn about the health consequences of tobacco use. The information gathered from and effects of tobacco company advertising will be discussed separately below. The major sources of information fall into a number of categories.

Health Care Providers

The influence of physicians and nurses as communicators of information and as exemplars of healthy life styles has been the subject of much research (198). The greater concern about health among women, and their greater contact with health professionals, provides an obvious avenue of intervention (114). Health professionals should be continuously reminded of their potential impact and advised to use it to influence women to reduce smoking. Physicians are considered the most authoritative source, with the greatest potential for influencing patient behavior.

From the self-report of adults, physicians are not delivering enough anti-smoking information and advice. In 1975, a full 64.6 percent of male and 60.8 percent of female current smokers claimed that they had never received advice from any doctor about quitting, cutting down, or continuing smoking (194). About 19 percent of male and 21 percent of current female smokers had been advised to quit. Combining advice to quit and/or cut down, the percentages rose to 34.8 percent of men and 37.7 percent of women. In 1970, the percentages of men and women who reported such advice were 30.2 percent and 34 percent, respectively (193). A somewhat lower estimate of physician advice was obtained from an ongoing nationwide study involving approximately 8,000 people (184). Advice to quit or cut down was reported by 22.4 percent of the subjects, and lack of advice by 77.6 percent; there were no significant gender differences.

A survey of physicians' opinions about smoking and health in the mid-1960s revealed that 38 percent claimed they advised "all" or "almost all" (95 to 100 percent) of their patients who did not have smoking-related disorders to quit or cut down (76). Eighty-eight percent of physicians claimed they gave such advice to patients with lung and pulmonary conditions.

Nurses spend more time in direct patient contact than do physicians and can exert a major role in delivering information as well as serving as exemplars. Most nurses are aware of this responsibility (60,75,135,195). Only 10 percent of nurses claimed to discuss smoking and health with "almost all" or "most" (65 to 99 percent) of their patients or students (135). Another 21.5 percent claimed to have discussed it with 35 to 64 percent of patients or students. Only 50 percent of current smokers, compared to 65 percent each of former smokers and nonsmokers, suggested stopping to 5 percent or more of their patients and students.

While the identical question was not asked of nurses in the 1975 survey, a number of valuable questions relating to exemplar status were posed (196). In almost every case, current smokers took the weakest position on exemplar role, former smokers were in between, and nonsmokers were strongest. For all questions, the proportion of nurses who agreed "strongly" or "somewhat" with the statements of exemplar role is reported here. Regarding their own behavior, 69.5, 91.7, and 94.5 percent of current, former and nonsmoking nurses respectively felt that they should set a good example by not smoking. This percentage varied according to work location. Lowest percentages were given for hospital duty (70.0, 83.3, and 89.2 percent for current, former and nonsmokers respectively), intermediate for private physician's office (79.9, 86.7, and 90.5 percent, respectively, and highest for private duty (91.1, 91.4, and 94.4 percent, respectively). A much lower rate of agreement about not smoking in public while in uniform was obtained; only 44.4 percent of current smokers, 67.1 percent of former smokers, and 72.8 percent of nonsmoking nurses concurred. Nurses believe that it is their responsibility to convince people to stop smoking (64 percent of smokers, 74 percent of former smokers, and 64.8 percent of nonsmokers). Approximately 54 percent of smokers, 81.3 percent of former smokers, and 82 percent of nonsmokers said they had tried to persuade someone other than patients to quit, and a much higher percentage reported convincing someone not to start (83.4, 78.6, and 75.8 percent, respectively). Finally, 52.1, 78.2, and 85.4 percent of the respective groups agreed strongly or somewhat that nurses should be more active in speaking to lay groups.

Given the possible role modeling effect of female nurses, a need exists for adequate preparation of all health professionals in smoking and health counseling. This preparation should include education on the health hazards of smoking as well as effective methods of counseling patients.

There is little information available about the role played by other health care providers in dissemination of information or discouragement of smoking behavior. Nationwide campaigns are currently being aimed at physicians and dentists to increase their commitment to and involvement with this task. Other health care providers should be encouraged to take a more active role and adopt exemplar status as well.

Educators

Adult educators include those in schools and colleges, job training, community organizations (churches and other religious groups, Young Women's Christian Associations, and Red Cross, civic organizations, social service groups, cultural groups) and in school-based programs for parents. There are large number of sources of information about smoking available from educators in adult settings and in programs for parents. These have been studied in-depth and reviewed elsewhere (188, 198). The frequent contact with and involvement of women in the school system should provide excellent opportunities to provide female-oriented information.

Peer Group

This group is an important, influential source of information on behavior. Evidence is strongest for the effect on initiation (addressed earlier in this Part). In two studies of British working class women, the peer group was an important source of information about smoking and pregnancy (11,74). Other strong relationships within the lay adult community have also been reported (118,201).

Family

Significant others, especially within the family, have been shown to be primary sources of information to pregnant women (11,74). The female relative may serve as a particularly important role model for black women (201). Smoking initiation is strongly influenced by parental smoking habits in teenagers (addressed earlier in this Part). In married couples, smoking patterns tend to be congruent; this almost enforces a sharing of information and makes it especially important in quitting efforts that couples stop together or are very supportive of the new ex-smoker (77,118,170,216).

Media: Television, Radio, Film, Newspapers, Magazines

The use of the mass media as a source of information as well as a tool in effecting cessation has been extensively developed in recent years (55,188,193,198,202,214).

Since women are almost exclusively the target audience of women's service magazines, effort should be devoted to using this medium to provide information on smoking and health, cessation techniques, and clinic availability. These magazines have not adequately disseminated information on smoking and health.

One of the principal reasons suggested for this failure is the power that tobacco companies wield through the economic incentive of advertising (178). Only one women's service magazine does not accept cigarette advertising in the United States. Frank admission of the economic dependency upon such advertising has been made. Not a single leading national woman's magazine that accepts cigarette advertising in 7 years of publication printed an article "... that would have given readers any clear notion of the nature and extent of the medical and social havoc being wreaked by the cigarette-smoking habit" (178). Smith goes on to point out that those magazines that do not accept cigarette advertising, or have no advertising at all, have done considerably better at informing their readers of the health risks of smoking.

Advertising

In recent years, advertising in the United States has been directed specifically towards the women's market, with themes as diverse as the emancipation of women, the first woman (biblical reference), romantic love, and the independent single woman. Most girl smokers have a positive impression of the individuals pictured in cigarette advertisements. The latter are seen as attractive (by 69 percent), enjoying themselves (by 66 percent), well dressed (by 66 percent), sexy (by 54 percent), young (by 50 percent), and healthy (by 49 percent). There is no comparable data on how girl nonsmokers or young adult women view advertising (216).

Thus, advertisers have been successful in creating a sense of mystery, sophistication, and power around the behavior of smoking. Although smoking was once frowned upon for women, people now respond less negatively to a woman smoking (16). There is evidence that, for some women, smoking is linked with attitudes and behaviors that comprise a socially valued and successful self-image, and that giving up smoking is a threat to that image (123).

A majority of former smokers and nonsmokers of both sexes in the 1975 Adult Use of Tobacco Survey (194) agreed with the statement, "Cigarette advertising should be stopped completely." The percentages for men were 56.9 percent for nonsmokers and 56.4 percent for former smokers, and for women, 68.2 percent for nonsmokers, and 62.5 percent for former smokers. However, only 42.6 percent of male smokers and 42.5 percent of females smokers agreed with the statement. It appears that adult smokers value cigarette advertisements, but why they do—whether for information about brand characterization and availability, identification with the image portrayed, or some other reason—is not known. Fishbein concluded that cigarette advertising influences the decision to smoke as well as the choice of brand. Furthermore, he points out that cigarette advertising may serve as a discriminative stimulus for smoking behavior. Advertising can influence the initiation of smoking, the choice of brands smoked, and the level of consumption. Commenting that the tobacco industry asserts that advertising serves only to influence brand choice and not initiation or consumption, Fishbein maintains that it is somewhat unrealistic to assume that an advertisement which can do one of these things is not also capable of doing the other. While additional research on the effects of cigarette advertising is clearly necessary, this review suggests that cigarette advertising does affect cigarette consumption (63).

Restrictions have now been placed on advertising in many countries in the world, including the United States. There is no uniform agreement that the ban on televised cigarette advertising in the United States and the United Kingdom significantly reduced consumption. However, it is generally believed that each action of this sort—including the U.S. Surgeon General's Reports and the Reports of the Royal College of Physicians, as well as other smoking control measures such as taxation and legislation—has a cumulative effect on per capita consumption (8,142,202).

THE FAILURE TO DISSEMINATE INFORMATION

Many of the critical evaluations of public health campaigns conveying anti-smoking information maintain that little attitudinal or behavioral change is ever effected (188). Fishbein (63) argues that there is insufficient information describing the complex relationships between cigarette smoking behavior and beliefs, attitudes, and intentions to make this conclusion. He further maintains that it is necessary to know to what extent decisions regarding initiation, reduction, increase or cessation are under attitudinal (individual, personal) or normative (society-influenced) control. The importance of personalizing the health message, and the failure of the public to personalize the health messages that they have received is emphasized. For example, over 80 percent of smokers agree with the statement that smoking is hazardous to health. However, on the question, "Are you in any way concerned about the possible effects of cigarette smoking on your health?" only 25 percent of current smokers in the 1975 NCSH survey stated that they were "very concerned," another 22.6 percent were "fairly concerned," 18.9 percent were "only slightly concerned," and a final 31.9 percent were "not concerned" (194). Fishbein maintains that the public is not effectively informed about the general danger to health posed by smoking and is even less informed about the connection with specific diseases. He concludes that the content of an effective message is fourfold: that continued smoking leads to negative outcomes; that stopping smoking leads to positive outcomes; that personal relevance must be established; and that normative influences must be appealed to by maintaining that significant others think an individual should quit.

Stress at Work

A general model of stress at work (38) is worthy of consideration. Examination of the sources of stress at work (Figure 2) reveals a number of items that are especially salient for women. Discrimination against women in employment, role conflict, authority problems, inequity in promotions, exclusion from decision-making processes and the "old boys" network have been frequently discussed (68). Individual characteristics may be considered from a gender viewpoint as well; for example, some types of psychological disorders, such as anxiety and de-

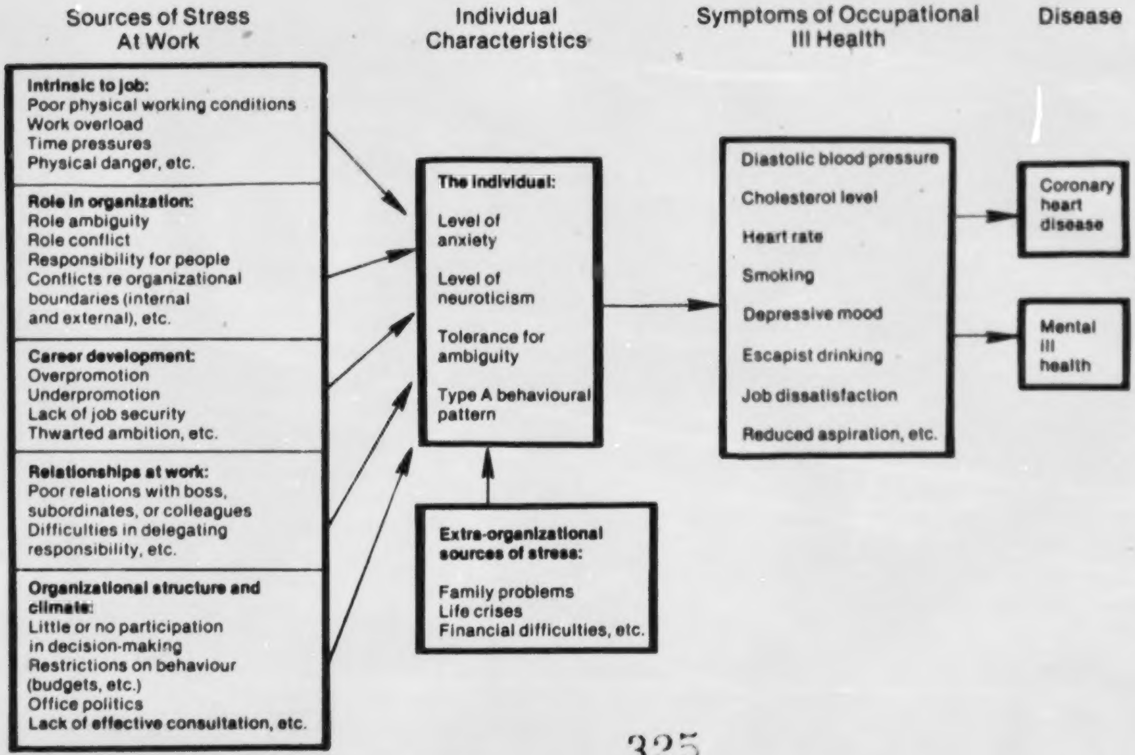
Sources of Stress At Work
Individual Characteristics
Symptoms of Occupational Ill Health
Disease
Intrinsic to job:
Poor physical working conditions
Work overload
Time pressures
Physical danger, etc.
Role in organization:
Role ambiguity
Role conflict
Responsibility for people
Conflicts re organizational boundaries (internal and external), etc.
Career development:
Overpromotion
Underpromotion
Lack of job security
Thwarted ambition, etc.
Relationships at work:
Poor relations with boss, subordinates, or colleagues
Difficulties in delegating responsibility, etc.
Organizational structure and climate:
Little or no participation in decision-making
Restrictions on behaviour (budgets, etc.)
Office politics
Lack of effective consultation, etc.
The individual:
Level of anxiety
Level of neuroticism
Tolerance for ambiguity
Type A behavioural pattern
Extra-organizational sources of stress:
Family problems
Life crises
Financial difficulties, etc.
Diastolic blood pressure
Cholesterol level
Heart rate
Smoking
Depressive mood
Escapist drinking
Job dissatisfaction
Reduced aspiration, etc.
Coronary heart disease
Mental ill health

FIGURE 2.—A model of stress at work

Items marked by asterisk (*) are particularly relevant to *female workers.
SOURCE: Cooper, C.L. (38).

pression, are more prevalent among women than men (48,68). The Type A behavior pattern, which is associated with male cardiovascular disease, has been shown to be unrelated to sex once socioeconomic status is taken into consideration (172).

An additional set of stressors originates in the extraorganizational environment. A prospective study of the relationship of employment status and employment-related behaviors to coronary heart disease (CHD) incidence was conducted by Haynes and Feinleib (91). Working women scored higher on scales measuring daily stress, marital dissatisfaction, and aging worries than men. They were also less likely to display overt anger than either homemakers or men. While incidence rates of coronary heart disease in working women were not significantly higher than in homemakers, an excess risk of CHD was identified among women who were employed in clerical jobs and had children. The risk factors for CHD in this group included family responsibilities, suppressed hostility, a nonsupportive supervisor, and low job mobility over the preceding 10-year period.

Smoking Habits of Health Professionals

There are relatively few studies available which present gender-specific smoking rates in various professions. Health professionals were selected for analysis because they were more likely to be aware of the health consequences of smoking than the general public; this group has also been studied more extensively.

PHYSICIANS

The smoking habits of male and female physicians in five nations are presented in Table 15. Smoking rates in the general population are provided for comparison when supplied by the authors. No breakdowns by gender are available for the United States. Separate estimates of smoking rate in a small group of women physicians age 36 to 46 at the time of survey (205) and in a large sample of predominantly male (93 percent) physicians (195) are listed in the table. In addition, the wives of 3,990 physicians were queried about their own smoking habits and those of their husbands; no information is provided on the occupations of these women (77).

Examination of the table shows that smoking rates of physicians, both male and female, tend to be much lower than general population rates. The only exception is the higher rate of current smokers among female physicians in Finland (200). The percentage of current smokers among the sample of U.S. female physicians is higher than that reported in other

TABLE 15.—Smoking habits of male and female physicians in selected countries

			Percent Smokers					
Author	Country	Number	Never	Pop. Est.	Current	Pop. Est.	Former	Pop. Est.
1. Bourke, et al., 1972 (22)	Ireland	M 1359	17.9 —	19.7*	48.5 —	67.6*	33.6 —	12.7*
		F 221	51.5	53.9	26.7 —	38.6*	22.2 —	7.5*
2. Vuori et al., 1971 (200)	Finland	M } 843	38		34	60	27	
		F	66		26	20	8	
3. Wilhelmsen & Faith-Ell, 1974 (210)	Sweden	?	33		38		29	
			54		27		19	
4. Aaro et al., 1977 (1)	Norway	M 740			35.3 —	53*	37[1] —	27[1]*
		F 398			21.7 —	36*	38[1] —	20[1]*
5. Westling-Wikstrand et al., 1970 (205)	USA	F 81	42		35.8		13.6	
6. Greenwald et al., 1971[2] (77)	USA	M 3990	32[3]		24		43[3]	
		F 3990	35[3]		36		27[3]	
7. USDHEW, 1976 (195)	USA	M 3657[4]		21	39	64[1]	43[1]	34[1]

*Significant difference between percentages paired by (—).

[1] Stopping rate $= \frac{\text{former smoker}}{\text{ever smoker}}$

[2] Sample consisted of physicians and their wives whose profession was undefined.

[3] Percentages estimated from graph, not specified in text.

[4] Approximate total of M and F, estimated to be 93% male.

countries and approaches the rates in the general population (205). Prevalence of smoking has a strong relation to demographic variables such as profession, income, and education. We would expect physicians to be in the highest category on each of these variables and, therefore, to have lower prevalence rates. Therefore, it would be relevant to examine the cross-tabulations for smoking prevalence by socioeconomic status, according to sex.

According to the three studies providing comparative data, both female and male physicians are quitting at rates higher than the general population. The percentage of former smokers among female physicians, and estimates of quit rate, are lower than among male physicians in all but one of the studies listed. This trend may represent a time lag in the smoking behavior of women as compared to that of men, or there may be a lower quit rate among women physicians.

In two studies, female physicians smoked more cigarettes per day than women in the general population (1,22). In contrast, wives of physicians smoked fewer cigarettes on the average than their husbands (77). A greater percentage of the wives of physicians than physicians themselves were smokers in every age group except the oldest. The percentage of current smokers appeared to be inversely related to age in the group of wives, but virtually stable across age for the physician-husbands. Husbands and wives tended to have similar smoking habits.

Based on a small sample of women graduates of a single U.S. medical school, Westling-Wikstrand, et al. (205) reported that 58.8 percent of the current smokers belonged to the category "professor" (academic appointment of assistant professor or above, with or without board attainment) when ranked on professional attainment. The other categories were "boards" (specialty board certification but not professional appointments), "no boards" (in practice without board certification or professional appointment), and "not in practice." The "professor" group was characterized by greater likelihood of being single and having fewer "habits of nervous tension." Compared to other groups, this group had the lowest depression scores, average anger scores, and the highest anxiety scores. The authors comment that this group of women was the most similar to their male colleagues. They may also have experienced fewer problems with ambivalence about sex roles, self-image, or conflict over aggressive behavioral patterns. The presence of the high anxiety scale, however, casts some doubt on this generalization.

Women in U.S. medical schools are subjected to significant psychological pressures and often experience emotional problems and lack of confidence about achieving the goal of graduation (205). Female physicians also experience significant role conflict (19).

The relevance of indices of stress to smoking patterns is again one of inference. If smoking serves as a coping mechanism—a means of reducing negative affect—then it is understandable that female physicians, or any other professional with elevated stress levels, would have higher current smoking rates than the general populace. It is also understandable that they might experience more difficulty in quitting.

PSYCHOLOGISTS

A survey of psychologists in California state universities and colleges found that female psychologists were much more likely to smoke than their male colleagues (46). The rate of smoking was slightly higher than in male health professionals, and approximately the same for female psychologists (38 percent) and nurses (195) (see Table 16).

This smoking rate is significantly above the rate among professional women in general (25.6 percent) and was due to lower cessation rates among psychologists rather than higher initiation rates. The most common reasons given for smoking are the stress of work or school, and personal stress. Frieze, et al. state that professional women have to exhibit "male-like" characteristics in order to survive in their jobs, but that these characteristics are often met with criticism and hostility (67). Thus, social and occupational demands are at odds with each other. Furthermore, there is evidence that female psychologists face very real sex discrimination in the evaluation of their work (67).

Dicken and Bryson (46) report a high degree of power fantasies among female psychologists who smoke. This supports Fisher's finding that female smokers in general seem preoccupied with the issue of power (64). He speculates that cigarettes are used defensively against feelings of powerlessness, weakness, and inferiority.

Elevated suicide rates are another correlate to the evidence of excessive stress and difficulty in coping experienced by some female professionals. These higher rates, compared with the general female population, have been observed among women psychologists, chemists, and physicians (124,164). Factors such as ambivalence about success, role conflict and marginality were offered as dynamics. However, it is not possible to determine whether these higher suicide rates are due to the self-selection of suicide-prone women into these and possibly other professions, or to the difficulties encountered in professional training and practice (or to an interaction of both).

NURSES

A number of studies have shown a higher rate of smoking among nurses than in the general female population in the United States. The most recent assessment of nurses' smoking behavior was conducted in 1975 (199). In Table 16, smoking habits of nurses are compared with those of adult U.S. women and other groups of health professionals.

Between 1969 and 1975, the proportion of nurses who were current smokers rose from 37 to 39 percent. Every other category of health professional (physician, dentist, and pharmacist) had substantially reduced smoking rates. The membership of these three professions is predominantly male and current smoking rates vary from 21 to 28 percent. If one examines quit rates in 1975 among the four categories of health professionals, it is clear that the majority of physicians, dentists, and pharmacists who ever smoked cigarettes have quit: 64, 61, and 55 percent respectively. Among nurses, only 36 percent have quit, which does, however, compare favorably with adult women (34 percent) and working women (30 percent) (199).

Noll surveyed smoking behaviors of nurses by work setting (see Table 17) (135). The overall percentage of current smokers in this survey was 37 percent, compared to a national average (for 1966) of 33.7 percent in women. There was a smaller percentage of never smokers (41.3 percent) among nurses in that survey than among the female population (56.8 percent), suggesting a higher quitting rate at that time as well. From Table 17 it appears that there is no selective recruitment into the various nursing specialties; the proportion of never smokers is fairly equal across work settings. Differences do appear, however, in the proportion of current smokers according to work setting. Highest rates of smoking are found in psychiatric and pediatric settings, and lowest rates in the four categories connected to education and community involvement: nursing education, working in the community, elementary or high school nursing, and working in a doctor's office.

In Great Britain, only 26 percent of maternity nurses smoked regularly, compared to 37 percent of those in general nursing (106). In the United Kingdom, approximately the same proportion of nurses smoke as women in the general population —44 percent (106,154).

Knopf Elkind reports differences in smoking among different types of ward nursing staff. Trained nurses had 41 percent current smokers, learners had 28 percent, nursery nurses had 14 percent, and auxiliaries had 61 percent current smokers (106).

Lampman reported a similar excess of smokers among nurses

TABLE 16.—Percentages of cigarette smokers (S), former smokers (FS), and ever smokers (ES) and cessation ratio (FS/ES) among psychologists, nurses, and other selected health professionals

Sample	N	S	FS	ES	FS/ES
Male and predominantly male samples					
CSUC male psychologists	258	28	35	62	55
Eminent experimental psychologists—90% male (Lawton and Goldman, 1961)	72	53	11	64	17
Psychiatrists —% male not reported (Tamarin and Eisinger, 1972)	309	42	27	69	39
American Public Health Association male members (Eyres, 1973)	3,569	21	40	61	66
Physicians—93% male (USPHS, 1977)	3,657	21	42	63	67
U.S. adult males (USDHEW, 1976)	5,702	39	29	69	42
Female and predominantly female samples					
CSUC female psychologists	86	38	19	57	33
American Public Health Association female members (Eyres, 1973)	1,973	31	31	62	50
Nurses—98% female (USPHS, 1977)	2,429	39	22	61	36
U.S. adult females (USDHEW, 1976)	6,327	29	14	43	33

NOTE: CSUC = California State University and Colleges.
SOURCE: Dicken, C. (46).

TABLE 17.—Cigarette smoking status by work setting for nurses (percent)

Work Setting	Cigarette Smoking Status			Total*	
	Current	Former	Never	Percent	N
Surgical Units	41.2	19.4	39.4	100.0	529
Medical Units	37.8	18.2	43.9	99.9	476
Operating, Labor, Delivery Emergency Room	39.8	15.2	45.0	100.0	485
Maternity Unit	36.2	17.2	46.6	100.0	197
Pediatrics Unit or Setting	46.6	8.8	44.6	100.0	80
Psychiatric Unit or Setting	49.9	18.2	32.0	100.1	135
Nursing Education Setting	24.6	26.8	48.7	100.1	90
In the Community	26.1	33.4	40.6	100.1	264
Elementary or High School	27.5	36.4	36.1	100.0	217
Doctor's Office	24.2	33.8	41.9	99.9	338
Out-Patient Clinic	42.5	15.1	42.5	100.1	113
Other and Mixed	41.3	18.4	40.3	100.0	1,078

*Total N = 6,012
SOURCE: Noll, C.E. (135).

aides (95.2 percent female) in a large metropolitan hospital in the United States (110). Fifty-two percent of that group smoked, compared with 36 percent of the medical nurses (99.3 percent female) and 40 percent of the student nurses (95.6 percent female). This survey was aimed at identifying smoking within the hospital. Thus, true prevalence in this sample can only be higher.

Compared to other female health professionals (see Table 16) in the United States, nurses' quit rates are above some (psychologists, U.S. adult women) and below others (American Public Health Association female members). Knopf Elkind points out that in the British population other female-dominated professions, such as primary school teachers, health visitors and domiciliary midwives, have noticeably lower rates of smoking than hospital nurses (106). Entry into the profession of nursing is associated with taking up daily smoking but the degree of occupational stress in a population of 300 British student nurses was not different for smokers and nonsmokers (92). This finding does not rule out the use of smoking as a stress-reduction mechanism, however.

Other factors which might contribute to a high smoking rate among nurses are work overload and frustration in professional relationships with physicians.

Knowledge of health consequences of smoking is high among nurses, but it has been shown that student nurses are less well-informed than medical students (154). Nurses who quit smoking do cite protection of future health as a major reason (75,92). Nurses who smoke are less likely than nonsmokers to agree that not smoking is a preventive measure against cancer (106). Similar refusal to acknowledge health risks of smoking is found among smokers in the general population (194). Whether this represents a real lack of knowledge or a method of reducing cognitive dissonance through denial is unknown. The problem is particularly critical for nurses (and other health professionals) since they serve both as exemplars and as providers of information (106).

The Pregnant Smoker—a Special Target

The pregnant woman is in a unique life situation. Every substance she ingests and every behavior that she manifests can affect the present and future health status of the fetus she is carrying. If she smokes, the nicotine, carbon monoxide, and hydrogen cyanide which she inhales all cross the placental barrier and enter the bloodstream of the fetus. The risk factors for both mother and fetus have been extensively reviewed elsewhere in this volume as well as in previous reports from the Surgeon General (198). (See also Pregnancy and Infant Health in Part II of this Report).

It is estimated that between one-quarter and one-third of pregnant smokers quit smoking for the duration of pregnancy and that another third cut down.

This section reviews the current literature on sources of information available to the pregnant smoker, summarizes available data on prevalence of current smoking and smoking cessation during pregnancy, and discusses the problem of cessation from a behavioral viewpoint.

SOURCES OF INFORMATION

The same classes of information discussed in the previous section are available to the pregnant smoker. How the pregnant smoker uses these sources and her degree of confidence in the information provided seems to be a function of socioeconomic status and parity. Information is distributed through health professionals (primarily physicians and nurses), peers and family, community resources, and the media.

Women in lower socioeconomic classes tend to rely more on lay referral systems, such as peers and family, than upon mass media or medical sources (10,74). Personal transmission of information seems to be more highly valued and readily adhered to (71). Middle and upper class women are more likely to utilize impersonal sources such as mass media and physician-supplied information (74).

In one study of predominantly working class British women, the mode of exposure to smoking information ranked as follows: 84 percent had seen it on television; 65 percent were told by family or friends; 52 percent had seen posters and leaflets; 37 percent had been told by husbands; 34 percent used books and magazines; and 25 percent had been told by a medical source (16 percent from a doctor, and 9 percent from a nurse) (11). The authors comment that television, posters, and leaflets are inadequate for the delivery of statistical information; books, which are better sources, were used much less than these other sources. Baric and MacArthur present a discussion of health norms in pregnancy (10). Seventy-nine percent of the sample were aware of some norm relating to smoking in pregnancy: 39 percent thought they were expected not to smoke at all, and an additional 40 percent thought they were expected to reduce their smoking. All of the women could name at least one source of information; 98 percent had been exposed to mass-media messages to quit smoking. Smoking seemed to be undergoing a change in norm status, from generality to specificity, i.e., from being a general health menace to one with specific consequences, such as a threat to the health of the baby.

The issue of normative behavior in smoking and personalization of message should be crucial to informational campaigns, according to Fishbein's theory (63). Social support from a spouse should also be critical, as would be involvement of significant others.

Women about to have their first baby are more likely to believe educational materials than multiparous women (11,50). This finding suggests that different modes of intervention or different emphases should be developed for primiparous and multiparous women.

Physician Advice

The physician represents one of the most knowledgeable figures the pregnant woman will encounter as a source of information. Consequently, estimates of the frequency with which the physician delivers advice on smoking are of importance.

Three such estimates are available from national samples in the United States. In the first study, conducted in the mid-1960s, 37 percent of physicians reported that they advised all or almost all (95 to 100 percent) of their pregnant patients to quit smoking or cut down sharply. Obstetricians were more likely to deliver such advice to pregnant patients (49 percent) than were physicians in general practice (38 percent) (76).

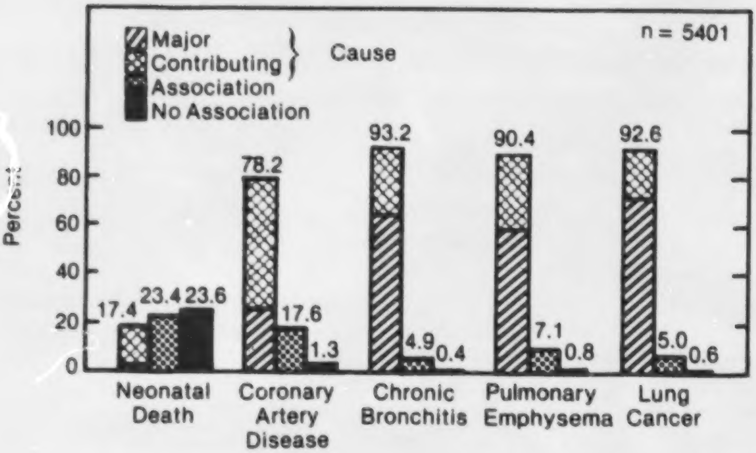

FIGURE 3.—Beliefs of OB-GYN specialists about the association of maternal smoking with neonatal death and other selected diseases

SOURCE: Danaher, B.G. (40).

The Physician Advice Survey conducted by the Center for Disease Control examined the beliefs and behavior of physicians specializing in Obstetrics and Gynecology (OB-GYN) in the United States (40). The OB-GYN specialty practice includes preventive medical care in the form of specific suggestions regarding hygiene and family planning and, during pregnancy, active participation in directing perinatal care (40). The beliefs of OB-GYN specialists about the relationship between maternal smoking and neonatal death are presented in Figure 3, along with their belief about some of the more common diseases associated with smoking. Because neonatal death can result from a great many factors, the attribution of causality is somewhat lower than for the other conditions represented. However, it is notable that 23.6 percent of the physicians deny the existence of any relationship. Congruent with the estimate from the 1960s, 45.3 percent of OB-GYN specialists in this survey claimed to instruct all or almost all of their patients to quit or cut down on smoking (see Figure 4). Another 13.1 percent delivered such advice to most or many (65 to 95 percent). A noticeably smaller fraction of physicians who are current smokers deliver this message than ex-smokers or nonsmokers.

The 1975 Survey of Adult Use of Tobacco, sponsored by the National Clearinghouse on Smoking and Health, included a

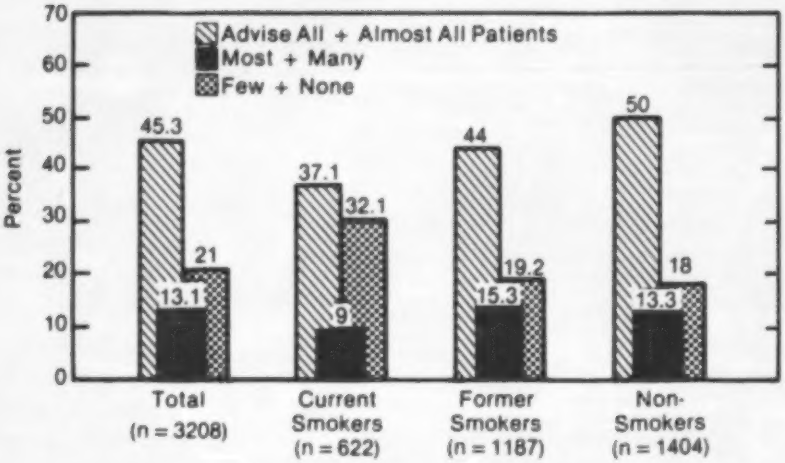

FIGURE 4.—Percentage of patients advised to quit or cut down their smoking by the smoking behavior of the advising obstetrician-gynecologist

SOURCE: Danaher, B.G. (40).

questionnaire directed at smoking habits in pregnant women. A preliminary analysis of the results has been made (89). Out of 12,029 respondents interviewed in 1975, a total of 1,225 women (814 current smokers and 411 former smokers) were administered questions about their smoking habits during pregnancy.

Each of the 983 respondents (664 current smokers and 319 former smokers) who had ever been pregnant was asked whether her doctor suggested that she quit smoking or cut down during her last pregnancy. Table 18 displays the results by year of last pregnancy. The percentage of women reporting such advice from their doctors rose steadily. Only 14.6 percent of women who had last been pregnant from 1965 to 1969 claimed to have been advised by their doctor either to stop or cut down; 23.7 percent of women last pregnant from 1970 to 1975 remembered such advice. These estimates are considerably smaller than those supplied by physicians themselves (40,76). There are several possible explanations for the discrepancy: the women were reporting retrospectively, and memory may have been distorted; a selective under-reporting of advice may have occurred; or the populations of physicians and patients may be entirely nonoverlapping. Retrospective data have been shown to be unreliable in one pregnancy study (49). Unfortunately, sample sizes were too small to provide reliable estimates of the per-

TABLE 18.—Distribution of responses of current/former smokers who were ever pregnant to the question "Did your doctor suggest that you cut down or stop smoking cigarettes during your last pregnancy?"

Physician's Advice	Percent by Year of Last Pregnancy (Prior to 1965)	(1965-69)	(1970-75)	(1965-75)
Quit smoking	5.6	6.2	10.8	9.3
Cut down smoking	5.7	8.4	12.9	11.4
No advice given	70.5	64.1	65.6	65.1
Not smoking at the time	16.4	20.6	9.1	12.9
Had no doctor	0.5	0	0.2	0.1
Don't know or no answer	1.3	0.8	1.3	0.9
Number of respondents	466	215	291	506

SOURCE: National Clearinghouse for Smoking and Health (194).

centage of women who followed the advice of a physician to stop smoking during pregnancy. Such data might have yielded an estimate of the effectiveness of such advice.

In sum, over 50 percent of physicians claim to advise their pregnant patients to eliminate or sharply curtail their smoking during pregnancy, but a much smaller percentage of pregnant women recall such advice.

PREVALENCE OF SMOKING AND QUITTING DURING PREGNANCY

The prevalence of smoking in pregnant women (before special cessation efforts) should be roughly equivalent to the prevalence of smoking in the female population in the same age range, corrected for socioeconomic status. Ten studies conducted in developed countries, reported between 1971 and 1973, show a range from 23.4 percent to 47.6 percent in prevalence of tobacco use (145). The median rate is 42.75 percent smokers for the entire sample. A survey (conducted during the course of the pregnancy) of 9,553 pregnant women who represent a cross section of the general population in the Riverside-San Bernadino-Ontario (California) area was recently completed (108). Preliminary results indicate that 44.5 percent of all women surveyed either continued to smoke during pregnancy or had smoked before, but not during, this pregnancy. Since the precise time of cessation is not clear, a more conservative estimate is that 33.3 percent of women continued to smoke for the duration of their pregnancy. This estimate is well within the range of those derived from the Population Report analysis (145).

There is a paucity of race-specific information on smoking prevalence during pregnancy. Niswander and Gordon (134), in a study encompassing 14 U.S. cities, reported greater prevalence of smoking among white than black women (53.65 percent vs. 41.85 percent, respectively). This is a high estimate and reversal of the prevalence rates presented in Table 7. The finding is similar to the previously presented data, in that white women smoked more cigarettes per day than black women: only 3.3 percent of black women smokers consume a pack a day or more, compared to 13.4 percent of white women in this study. Smoking is slightly less prevalent in black than in white women in the sample of Kuzma and Phillips (108): 57.3 percent of black women and 53.3 percent of white women have never smoked. For Hispanic women, the percentage is somewhat higher, 61.9 percent never-smokers. Table 19 summarizes the results of 11 studies reporting rates of discontinuing smoking during pregnancy. The overall rate of cessation among regular smokers ranges from 0.9 percent to 35 percent, which is the figure most often anecdotally cited. The median is closer to 20 percent.

Only one study provides ethnic data on smoking cessation during pregnancy (108). In this study, it should be remembered, stopped smokers are women who smoked prior to, but not during the pregnancy, so that quitting may not have been pregnancy-specific. Rates are very similar for white, black and Hispanic women: 24.5 percent, 24.9 percent and 28.7 percent, respectively, were stopped smokers in this study.

Even acute abstinence from cigarette smoking may be of value, if it occurs immediately prior to giving birth. In the United Kingdom, women are often admitted as early as 48 hours before elective delivery; abstaining from smoking for that period of time was found to result in a net percent increase in available oxygen as COHb was excreted (42). Such a temporary benefit may actually be critical under acutely stressful conditions, and where there is chronic placental insufficiency.

Cutting down on smoking during pregnancy would appear to be better than no change in behavior, especially for those adverse effects upon the fetus which show a dose-response relationship. However, cutting down on number of cigarettes does not always imply a reduction in delivered dose of nicotine or other tobacco smoke constituents (79,80). When smoking behavior was measured over the course of pregnancy in regular smokers (5 to 30 cigarettes per day for at least 5 years), a decrease in number of puffs per cigarette occurred as pregnancy progressed (6). Like puffing rate, the COHb concentration also decreased over time in pregnancy. However, in these subjects there was no significant change in nicotine dose extracted from

TABLE 19.—Percentage of current smokers who altered smoking behavior during pregnancy

		Change in Smoking Habit—Percent of Women					
Author and Date	N	Quit	Quit Temporarily	Cut Down Only	Increased	No Change	Miscellaneous, or Comment
1. Kullander & Kallen, 1971 (107)	2,806	0.9	1.3			97.3	+0.5 Initiated
2. Andrews & McGarry, 1972 (4)	6,733	14.7					Maternities only
3. Butler et al., 1972 (29)	341	18.4					Quit by end of 4th month
4. Schwartz et al., 1972 (171)	1,188	31.0		10.0			
5. Baric et al., 1976[1]	134	14.9	3.0			82.1	Quit by 1st ante-natal visit
6. Graham, 1976 (74)	50	33.3*		33.3*		33.3	*1/3 quit or cut down; 1/3 cut down temporarily
7. Baric & MacArthur, 1977[1] (10)	133	22.5	6.0	33.1	5.3	26.3	+6.8 reduced temporarily
8. Donovan, 1977 (49)	959	12.5	5.6				
9. Yankelovich et al., 1977 (216)	?	35.0		32.0			

TABLE 19.—Percentage of current smokers who altered smoking behavior during pregnancy—Continued

		Change in Smoking Habit—Percent of Women					
Author and Date	N	Quit	Quit Temporarily	Cut Down Only	Increased	No Change	Miscellaneous, or Comment
10. Harris, 1979 (89)	409[2]	26.5		24.8	7.9	36.9	+3.9 changed brand or switched to filter cigarettes 82.2 of quitters resumed smoking after delivery
11. Kuzma & Phillips, 1979 (108)	4,249	25.1[3]					13.4 of quit smokers were again smoking at 1-5 mo. post-delivery

NOTE: [1] These two studies may be composed of overlapping samples.

[2] Of the 506 women in the NCSH survey whose last pregnancy occurred during 1965-75, 409 reported smoking either before or during pregnancy.

[3] Percent who smoked prior to, but not during this pregnancy, calculated as part of smoker sample.

the cigarette over the duration of the pregnancy. Some alteration in puffing pattern, presumably in inhalation, produced the compensation. Thus, caution must be exercised in the interpretation of "cutting down."

There is even less information available on the percentage of quit-smokers who return to smoking after delivery. Table 19 provides two extremely divergent estimates: 82.2 percent (89) and 13.4 percent (108). Because we are dealing with relatively small sample sizes, the reliability of such data is not very high. Much more information must be accumulated before any firm statements about recidivism can be made. Women who quit during pregnancy have an excellent opportunity to change a behavior for life, with benefits both to themselves and to their children (see Recommendations).

PSYCHOSOCIAL FACTORS IN QUITTING

Health reasons, primarily centering around preventing harm to the fetus, are most often given as reasons for quitting. Yankelovich, et al. (216) report that 62 percent of young women smokers believe that smoking can harm the fetus and norms against smoking have been discussed (10). The sickness experienced as a part of pregnancy can also be a reason to give up smoking (11). It has also been reported that women who smoke before pregnancy show a significantly increased incidence of appetite cravings and aversions, which may be associated with quitting (41).

A closely related aspect of maternal health is weight gain. Preventing excessive weight gain has even been given as a reason to continue smoking during pregnancy (50). Baric and MacArthur included control of weight gain as a norm during pregnancy; 24 percent of this sample expressed awareness of social expectations in this area (10). The issue of how much weight it is appropriate to gain in pregnancy varies according to time and culture, so the generality of this finding is unclear.

Little is known about problems in quitting during pregnancy. The role of cigarettes as stimulants or tension reducers may be altered during this period. Abstinence symptomatology has also not been documented.

A composite picture of the successful quitter has been drawn by Baric, et al. and also by Kuzma and Phillips (11,108). Baric, et al. list educational qualifications as being positively related to quitting, followed by sickness in early pregnancy. Other distinguishing characteristics are smoking fewer cigarettes before pregnancy (also see 49,171), having started smoking at an older age, having stopped previously for at least 6 months, having heard about harmful effects of smoking from more sources, firmly believing that smoking was harmful to the baby, and finally, being encouraged to stop or being joined in the cessation effort by their husbands (47,166).

Kuzma and Phillips identified a number of similar characteristics: higher educational level; greater family income; being married; being employed; more frequent church attendance; having a spouse who does not smoke; and no illicit drug use (106,108).

The characteristics described—advanced educational level, higher socioeconomic status, wider information base, belief in stopping for the sake of the fetus, and spousal support—all fit with a model of behavior change involving information, personalization, and social norms (63).

Three studies evaluate smoking cessation interventions for pregnant women (11,41,49). Tables 9 and 10 show reported abstinence figures for two studies. One study (11) showed no difference between intervention and control groups, and the second study (41) showed 50 percent abstinence at 9-month follow-up for those completing treatment (11,41). This latter result is very encouraging but is based on a very small sample in an affluent community where the aforementioned factors of educational level, high socioeconomic status and orientation toward professional advice are operative.

RECOMMENDATIONS

The preceding discussion has revealed a number of findings which may be useful in improving methods of reaching the pregnant woman and offering her cessation interventions.

1. Pregnant women seem to know that smoking is harmful to health, and most acknowledge that it can be directly harmful to the fetus. This information about the baby's health should be made as specific as possible, and the mother's own health should be intricately interwoven in the theme. Quitting is for the good of both mother and baby, not the baby alone. The harmful aspects of smoking and the benefits of not smoking must be equally emphasized.

2. Mass media, such as television and film, are particularly good avenues for portraying women of varying ethnicity in a number of geographical and socioeconomic settings. Because of gender identification it is important to utilize women as the transmitters of information and advice. Information should be dispensed by as many different sources of contact in the prenatal clinic (or doctor's office) as possible, not solely by the

physician. The awareness of various health professionals should be raised in this regard.

3. Social norms and lay referral systems should be used as part of information dissemination and modeling influences. This is particularly true for women of lower socioeconomic status. It is important to involve the father of the child in the normative belief system and in a direct supportive effort of quitting. This should be particularly timely in an era when more and more couples are experiencing pregnancy and birth as a two-person process.

4. Much more emphasis must be placed on permanent smoking cessation rather than just during the time of pregnancy. Positive aspects of remaining an ex-smoker include better health for the mother and child and the future impact of role modeling as the child grows.

Summary

1. The percentage of 17-18 year old women who smoke has shown a steady rise between 1968 and 1979. It now appears, however, that the increase in smoking prevalence among all 12-18 year old females has leveled off and begun to decline. Young women born after 1962 show a substantially reduced initiation of smoking and will probably have a much lower prevalence of smoking as adults.

2. Those young women who do begin to smoke are starting to smoke regularly at a younger age, with more than half of the male and female adolescents who begin to smoke starting before the 10th grade.

3. The earlier tobacco is used and the greater the number of cigarettes smoked per day, the less likely an attempt to quit will be successful.

4. The percentage of women smokers who smoke more than one pack per day is increasing.

5. Adolescent and adult women are more likely to use low "tar" and nicotine cigarettes, smoke fewer cigarettes per day and inhale less deeply than do men, but the difference between the sexes in these patterns of smoking is decreasing. Adolescent and adult black women are more likely to be smokers than their white peers, but they smoke fewer cigarettes per day.

6. Adolescents from low income families, single parent families, and families with lower parental educational levels are more likely to become smokers.

7. Female and male adolescents are more likely to begin smoking if a parent or older sibling also smokes.

8. Adolescent smokers associate with peers who smoke, and nonsmokers associate with nonsmoking peers.

9. Adolescent girls overestimate the percentage of their peers who smoke and they have a very positive image of the people in cigarette advertisements, but they are less likely than adolescent boys to see smoking as a social asset.

10. Adolescent girls who smoke tend to be more outgoing, but feel less able to influence their future.

11. Adolescents experience stress due to feelings of unattractiveness, incompetency in school achievement and personal relations, limited opportunity for personal growth and concern over future social and economic roles. This stress may be the common mechanism producing the increased rates of smoking in some groups.

12. The factors associated with successful quitting by adolescents of either sex are lower number of cigarettes smoked per day, higher educational aspirations and achievement, greater acceptance of the health risk of smoking, and having more nonsmokers among their friends.

13. It is possible that women and men modify their smoking in order to maintain a constant nicotine level.

14. Women are more likely than men to smoke in order to reduce stress.

15. Women at higher education and income levels are more likely to succeed in quitting. Additional factors associated with successful quitting are a strong commitment to change, the use of behavioral techniques and reliable social support for quitting. Women have been reported to show lower rates than men of successful cessation following organized cessation programs, a difference which is less apparent in those programs that include social support.

References

(1) AARO, L. E., BJARTVEIT, K., VELLAR, O. D., BERGLUND, E. L. Smoking habits among Norwegian doctors 1974. Scandinavian Journal of Social Medicine 5: 127-135, 1977.

(2) ABELSON, H. I., FISHBURNE, P. M., CISIN, I. National Survey on Drug Abuse: 1977. A Nationwide Study—Youth, Young Adults, and Older People. Department of Health, Education, and Welfare, Public Health Service, DHEW Publication No. (ADM) 78-618, 1977.

(3) ALLEGRANTE, J. P., O'ROURKE, T. W., TUNCALP, S. A multivariate analysis of selected variables on the development of subsequent youth smoking behavior. Journal of Drug Education 7(3): 237-248, 1977-1978.

(4) ANDREWS, J., MCGARRY, J. M. A community study of smoking in pregnancy. Journal of Obstetrics and Gynaecology of the British Commonwealth 79(12): 1057-1073, December 1972.

(5) ARVIDSSON, T. Views on smoking withdrawal. Experiences with

smoking withdrawal in Stockholm. Social-Medicinsk Tidskrift 2: 113-116, 1971.

(6) ASHTON, H. Effect of smoking on carboxyhaemoglobin level in pregnancy. British Medical Journal 1(6000): 42-43, January 3, 1976.

(7) ASHWELL, M., NORTH, W.R.S., MEADE T. W. Social class, smoking and obesity. British Medical Journal 2(6150): 1466-1467, November 25, 1978.

(8) ATKINSON, A. B., SKEGG, J. L. Control of smoking and price of cigarettes—a comment. British Journal of Preventive and Social Medicine 29: 45-48, 1974.

(9) BANKS, M. H., BEWLEY, B. R., BLAND, J. M., DEAN, J. R., POLLARD, J. Long-term study of smoking by secondary school children. Archives of Disease in Childhood 53: 12-19, 1978.

(10) BARIC, L., MACARTHUR, C. Health norms in pregnancy. British Journal of Preventive and Social Medicine 31: 30-38, 1977.

(11) BARIC, L., MACARTHUR C., SHERWOOD, M. A study of health education aspects of smoking in pregnancy. International Journal of Health Education, 19(2, Supplement): 1-17, April-June 1976.

(12) BARNES, G. E., FISHLINKSY, M. Stimulus intensity, modulation, smoking and craving for cigarettes. Addictive Diseases: An International Journal 2(3): 384-479, 1976.

(13) BERECZ, J. Modification of smoking behavior through self-administered punishment of imagined behavior: a new approach to aversion therapy. Journal of Consulting and Clinical Psychology 38(2): 244-250, 1972.

(14) BERGLUND, E. Tobacco Withdrawal Clinics: The Five-Day Plan, Final Report. Oslo, Norwegian Cancer Society, 1969, 67 pp.

(15) BEWLEY, B. R., BLAND, J. M. Academic performance and social factors related to cigarette smoking by school children. British Journal of Preventive and Social Medicine 31(1): 18-24, March 1977.

(16) BLEDA, P. R., BLEDA, S. E. Effects of sex and smoking on reactions to spatial invasion at a shopping mall. Journal of Social Psychology 104: 311-312, 1978.

(17) BLITZER, P. H., RIMM, A., GIEFER, E. E. The effect of cessation of smoking on body weight in 57,032 women: cross-sectional and longitudinal analyses. Journal of Chronic Disease 30: 415-429, 1977.

(18) BLOCK, J. H. Issues, problems, and pitfalls in assessing sex differences: a critical review of the psychology of sex differences. Merril-Palmer Quarterly 22(4), 1976.

(19) BLUESTONE, N. R. The future impact of women physicians on American medicine. American Journal of Public Health 68(8): 760-762, August 1978.

(20) BORLAND, B. R., RUDOLPH, J. P. Relative effects of low socioeconomic status, parent smoking and poor scholastic performance on smoking among high school students. Social Science and Medicine 9: 27-30, 1975.

(21) BOSSE, R., ROSE, C. Smoking cessation and sex role convergence. Journal of Health and Social Behavior 17: 53-61, March 1976.

(22) BOURKE, G. J., WILSON-DAVIS, K., THORNES, R. D. Smoking habits of the medical profession in the Republic of Ireland. American Journal of Public Health 62(4): 575-580, April 1972.

(23) BOZZETTI, L. P. Group psychotherapy with addicted smokers. Psychotherapy and Psychosomatics 20: 172-175, 1972.

(24) BRUNSWICK, A. F. Health and drug behavior: preliminary findings from a study of urban black adolescents. Addictive Diseases 3(2): 197-

214, 1977.

(25) BRUNSWICK, A. F. Health stability and change: a study of urban black youth. Part I: Degree and kind of change. Part II: Effects of drug use and unemployment. American Journal of Public Health (In press).

(26) BRUNSWICK, A. F., BOYLE, J. M. Patterns of drug involvement: developmental and secular influences on age at inititation. Youth and Society 11(2): 139-162, 1979.

(27) BURNS, B. H. Chronic chest disease, personality, and success in stopping cigarette smoking. British Journal of Preventive and Social Medicine 23(1): 23-27, February 1969.

(28) BURNUM, J. F. Outlook for treating patients with self-destructive habits. Annals of Internal Medicine 81(3): 387-393, September 1974.

(29) BUTLER, N. R., GOLDSTEIN, H., ROSS, E. M. Cigarette smoking in pregnancy: its influence on birth weight and perinatal mortality. British Medical Journal 2(5806): 127-130, April 1972.

(30) CHAPMAN, R. F., SMITH, J. W., LAYDEN, T. A. Elimination of cigarette smoking by punishment and self-management training. Behavioral Results and Therapy 9(3): 255-264, 1971.

(31) CHERRY, N., KIERNAN, K. Personality scores and smoking behavior. British Journal of Preventive and Social Medicine 30: 123-131, 1976.

(32) CHILMAN, C. S. Adolescent Sexuality in a Changing American Society: Social and Psychological Perspectives. Department of Health, Education, and Welfare, Public Health Service, DHEW Publication No. (NIH) 79-1426, 1979, 384 pp.

(33) CLAUSEN, J. A. Adolescent antecedents of cigarette smoking: data from the Oakland growth study. Social Science and Medicine 1: 357-382, 1968.

(34) COATES, T. J., PERRY, C. Multifactor risk reduction with children and adolescents: taking care of the heart in behavioral group therapy. In: Upper, D., Ross, S. (Editors). Behavior Group Therapy: An Annual Review. Champaign, Illinois, Research Press. (In press)

(35) COLEMAN, J. S. The Adolescent Society. New York, The Free Press, 1961.

(36) COLEMAN, J. S. Youth: Transition to Adulthood, Report of the Panel on Youth of the President's Science Advisory Committee. Chicago, The University of Chicago Press, 1974.

(37) COMSTOCK, G. W., STONE, R. Changes in body weight and subcutaneous fatness related to smoking habits. Archives of Environmental Health 24: 271-276, April 1972.

(38) COOPER, C. L., MARSHALL, J. Occupational sources of stress: a review of the literature relating to coronary heart disease and mental ill health. Journal of Occupatonal Psychology 49: 11-28, 1976.

(39) CREIGHTON, D. E., LEWIS, P. H. The effects of different cigarettes on human smoking patterns. In: Thornton, R. E. (Editor). Smoking Behavior. Edinburgh, Churchill Livingstone, 1978, pp. 289-300.

(40) DANAHER, B. G. OB-GYN intervention in helping smokers quit. In: Schwartz, J. L. (Editor). Progress in Smoking Cessation. International Conference on Smoking Cessation, June 21-23, 1978. New York, American Cancer Society, 1978, pp. 316-328.

(41) DANAHER, B. G., SHISSLAK, C. M., THOMPSON, C. B., FORD, J. D. A smoking cessation program for pregnant women: an exploratory study. American Journal of Public Health 68(9): 896-898, September 1978.

(42) DAVIES, J. H., LATTO, I. P., JONES, J. G., VEALE, A., WARDROP,

C. A. J. The effects of stopping smoking for 48 hours on oxygen availability from the blood: a study in pregnant women. British Medical Journal 2(6186): 355-356, August 1979.

(43) DELAHUNT, J., CURRAN, J. P. Effectiveness of negative practice and self-control techniques in reduction of smoking behavior. Journal of Consulting and Clinical Psychology 44(6): 1002-1007, December 1976.

(44) DELARUE, N. C. A study in smoking withdrawal. The Toronto Smoking Withdrawal Study Centre—description of activities. Canadian Journal of Public Health, Smoking and Health Supplement 64(2): S5-S19, March-April 1973.

(45) DICKEN, C. Sex roles, smoking, and smoking cessation. Journal of Health and Social Behavior 19(3): 324-334, September 1978.

(46) DICKEN, C., BRYSON, R. Psychology in action: The smoking of psychology. American Psychologist 33(5): 504-507, May 1978.

(47) DOHRENWEND, B. S., DOHRENWEND, D. P. (Editors). Stressful Life Events: Their Nature and Effect. New York, John Wiley, 1974.

(48) DOHRENWEND, B. P., DOHRENWEND, B. S. Sex differences and psychiatric disorders. American Journal of Sociology 81(6): 1447-1454, May 1976.

(49) DONOVAN, J. W. Randomized controlled trial of anti-smoking advice in pregnancy. British Journal of Preventive and Social Medicine 31: 6-12, 1977.

(50) DONOVAN, J. W., BURGESS, P. L., HOSSACK, C. M., YUDKIN, G. D. Routine advice against smoking in pregnancy. Journal of the Royal College of General Practitioners 25: 264-268, 1975.

(51) DOUVAN, E., ADELSON, J. The Adolescent Experience. New York, John Wiley & Sons, 1966.

(52) DRAGASTIN, S. E., ELDER, G. H. Adolescence in the Life Cycle: Psychosocial Change and Social Context. Washington, D.C., Hemisphere Publishing Corp., 1975.

(53) DUBREN, R. Evaluation of a televised stop-smoking clinic. Public Health Reports 92(1): 81-84, January-February 1977.

(54) DUDLEY, P. L., AICKEN, M., MARTIN, C. J. Cigarette smoking in a chest clinic population—psychophysiologic variables. Journal of Psychosomatic Research 21: 367-375, 1977.

(55) EISER, J. R., SUTTON, S. R., WOBER, M. Can television influence smoking? British Journal of Addiction 73(2): 215-219, June 1978.

(56) EISINGER, R. A. Psychosocial predictors of smoking recidivisim. Journal of Health and Social Behavior 12: 355-362, December 1971.

(57) ELGEROT, A. Note on sex differences in cigarette smoking as related to situational factors. Reports from the Department of Psychology, The University of Stockholm, No. 512, December 1977, 3 pp.

(58) EVANS, R. I. Smoking in children: developing a social-psychological strategy of deterrence. Preventive Medicine 5: 122-127, 1976.

(59) EVANS, R. I., ROZELLE, R. M., MITTLEMARK, M. B., HANSEN, W. B., BANE, A. L., HAVIS, J. Deterring the onset of smoking in children: knowledge of immediate physiological effects and coping with peer pressure, media pressure and parent modeling. Journal of Applied Social Psychology 8: 126-135, 1978.

(60) EYRES, S. J. Public health nursing section: report of the 1972 APHA smoking survey. American Journal of Public Health 63(10): 846-852, October 1973.

(61) FEDERAL TRADE COMMISSION. Report of "Tar" and Nicotine Content of the Smoke of 167 Varieties of Cigarettes, May 1978.

(62) FEE, W. M., BENSON, C. Group therapy: a review of 6 years' experi-

ence in a Scottish anti-smoking clinic. Community Medicine 126(26): 361-364, December 24, 1971.

(63) FISHBEIN, M. Consumer beliefs and behavior with respect to cigarette smoking: a critical analysis of the public literature. In: Federal Trade Commission. Report to Congress: Pursuant to the Public Health Cigarette Smoking Act. For the year 1976, May 1977, 113 pp.

(64) FISHER, J. Sex differences in smoking dynamics. Journal of Health and Social Behavior 17: 156-163, June 1976.

(65) FLETCHER, C., DOLL, R. A survey of doctors' attitudes to smoking. British Journal of Preventive and Social Medicine 23: 145-153, 1969.

(66) FRIEDMAN, G. D., SELTZER, C. C., SIEGELAUB, A. B., FELDMAN, R., COLLEN, M. F. Smoking among white, black and yellow men and women: Kaiser-Permanente multiphasic health examination data, 1964-1968. American Journal of Epidemiology 96(1): 23-25, 1972.

(67) FRIEND, P., KARLIN, R., GILES, H. Sex bias in the evaluation of journal articles: sexism in England. British Journal of Social and Clinical Psychology 18: 77-78, 1979.

(68) FRIEZE, I. H., PARSONS, J. E., JOHNSON, P. B., RUBLE, D. N., ZELLMAN, G. L. Women and Sex Roles. New York, W. W. Norton and Company, 1978, 444 pp.

(69) FRITH, C. D. Smoking behaviour and its relation to the smoker's immediate experience. British Journal of Social and Clinical Psychology 10(1): 73-78, February 1971.

(70) GILBERT, R. M. Coffee, tea and cigarette use. (Letter). Canadian Medical Association Journal 120: 522-524, March 1979.

(71) GLAUSER, S. C., GLAUSER, E. M., REIDENBERG, M. M., RUSY, B. F., TALLARIDA, R. J. Metabolic changes associated with the cessation of cigarette smoking. Archives of Environmental Health 20(3): 377-381, March 1970.

(72) GOLLEDGE, A. H. Influencing factors in anti-smoking clinics. The Medical Officer 114: 59-61, July 23, 1965.

(73) GORROD, J. W., JENNER, P. The metabolism of tobacco alkaloids. In: Hayes, W. J., Jr. (Editor). Essays in Toxicology, Volume 6. New York, Academic Press, 1975, pp. 35-78.

(74) GRAHAM, H. Smoking in pregnancy: the attitudes of expectant mothers. Social Science and Medicine 10: 399-405, 1976.

(75) GREEN, D. E. Nurses are kicking the habit. American Journal of Nursing 70(9): 1936-1938, September 1970.

(76) GREEN, D. E., HORN, D. Physicians' attitude toward their involvement in smoking problems of patients. Diseases of the Chest 54(3): 180-181, September 1968.

(77) GREENWALD, P., NELSON, D., GREENE, D. Smoking habits of physicians and their wives. New York State Journal of Medicine: 2096-2098, September 1971.

(78) GRIFFITHS, R. R., BIGELOW, G. E., LIEBSON, I. Facilitation of human tobacco self-administration by ethanol: a behavioral analysis. Journal of the Experimental Analysis of Behavior 25(3): 279-292, May 1976.

(79) GRITZ, E. R. Smoking behavior and tobacco abuse. In: Mello, N. K. (Editor). Advances in Substance Abuse, Volume 1. Greenwich, JAI Press, 1980, pp. 91-158.

(80) GRITZ, E. R., JARVIK, M. E. Nicotine and smoking. In: Iverson, L. L., Iverson, S. D., Snyder, S. H. (Editors). Handbook of Psychopharmacology, Volume 11. New York, Plenum Press, 1978, pp. 426-464.

(81) GRITZ, E. R., SIEGEL, R.K. Tobacco and smoking in animal and

human behavior. In: Davidson, R. S. (Editor). Modification of Pathological Behavior. New York, Gardner Press, 1979, pp. 419-476.

(82) GUILFORD, J. S. Sex differences between successful and unsuccessful abstainers from smoking. In: Zagona, S. V. (Editor). Studies and Issues in Smoking Behavior. Tucson, University of Arizona Press, 1967, pp. 95-102.

(83) GILFORD, J. Group treatment versus individual initiative in the cessation of smoking. Journal of Applied Psychology 56: 162-167, 1972.

(84) HALL, S. M., HAVASSY, B. The obese woman: causes, correlates and treatment. Professional Psychology. (In press)

(85) HAMBURG, B. A. Early adolescence: a specific and stressful stage of the life cycle. In: Coehlo, G. V., Hamburg, D. A., Adams, J. E. Coping and Adaptation. New York, Basic Books, Inc. 1974.

(86) HAMBURG, B. A., KRAEMER, H. C. JAHNKE, W. A hierarchy of drug use in adolescence: behavioral and attitudinal correlates of substantial drug use. American Journal of Psychiatry 132(11): 1155-1163, November 1975.

(87) HANDEL, S. Change in smoking habits in a general practice. Postgraduate Medical Journal 49: 679-681, October 1973.

(88) HANSON, H. M., IVESTER, C. A., MORTON, B. R. Nicotine self-administration in rats. In: Krasnegor, N. A. (Editor). Cigarette Smoking as a Dependence Process. NIDA Research Monograph No. 23. Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, January 1979, pp. 70-90.

(89) HARRIS, J. E. Smoking during pregnancy: preliminary results from the National Clearinghouse on Smoking and Health, 1975 Prevalence Data. September 1979.

(90) HAYNES, R. B. A critical review of the "determinants" of patient compliance with therapeutic regimens. In: Sackett, D. L., Haynes, R. B. (Editors). Compliance with Therapeutic Regimens. Workshop/ Symposium on Compliance with Therapeutic Regimens, McMaster University, 1974. Baltimore, Johns Hopkins University Press, 1976, pp. 9-25.

(91) HAYNES, S. G., FEINLEIB M. Women, work and coronary heart disease: prospective findings from the Framingham heart study. American Journal of Public Health 70(2): 133-141, February 1980.

(92) HILLIER, S. Nurses' smoking habits. Postgraduate Medical Journal 49(576): 693-694, October 1973.

(93) HUHTI, E., TAKALA, J., NUUTINEN, J., POUKKULA, A. Chronic respiratory disease in rural women. Annals of Clinical Research 10: 95-101, 1978.

(94) HUNT, W. A., BARNETT, L. W., and BRANCH, L. G. Relapse rates in addiction programs. Journal of Clinical Psychology 27(4): 455-456, October 1971.

(95) HUNTWORK, D., FERGUSON, L. W. Drug use and deviation from self-concept norms. Journal of Abnormal Child Psychology 5(1): 53-60, 1977.

(96) IKARD, F. F., TOMKINS, S. The experience of affect as a determinant of smoking behavior. A series of validity studies. Journal of Abnormal Psychology 81(2): 172-181, April 1973.

(97) JESSOR, R. Marijuana: a review of recent psychosocial research. In: Dupont, R. L., Goldstein, A., O'Donnell, J. A. (Editors). Handbook on Drug Abuse. Washington, D.C., U.S. Government Printing Office, 1978.

(98) JESSOR, R., JESSOR, S. L. Problem Behavior and Psychosocial Development: A Longitudinal Study of Youth. New York, Academic Press, 1977.

(99) JICK, H., PORTER, J., MORRISON, A. S. Relation between smoking and age of natural menopause. Lancet 1(8026): 1354-1355, June 1977.

(100) JOHNSON, B. D. The race, class, and irreversibility hypothesis: myths and research about heroin. In: Rittenhouse, J. D. (Editor). The Epidemiology of Heroin and Other Drugs. Department of Health, Education, and Welfare, Public Health Service, National Institute on Drug Abuse, December 1976, pp. 29-32.

(101) JOHNSTON, L. D., BACHMAN, J. G., O'MALLEY, P. M. Drug Use Among American High School Students 1975-1977. Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, DHEW Publication No. (ADM) 78-619, 1977, 238 pp.

(102) KANDEL, D. B. Convergences in prospective longitudinal surveys of drug use in normal populations. In: Kandel, D. B. Longitudinal Research on Drug Use: Empirical Findings and Methodological Issues. Washington, D.C., Hemisphere Publishing Corp., 1978, pp. 3-38.

(103) KANDEL, D. B., FAUST, R. Sequence and stages in patterns of adolescent drug use. Archives of General Psychiatry 32: 923-932, 1975.

(104) KANZLER, M., JAFFE, J., ZEIDENBERG, P. Long and short-term effectiveness of a large-scale proprietary smoking cessation program—a 4 year follow-up of Smokenders participants. Journal of Clinical Psychology 32(3): 551-569, July 1976.

(105) KEUTZER, C. S. Behavior modification of smoking: the experimental investigation of diverse techniques. Behavior Research and Therapy 6(2): 137-157, May 1968.

(106) KNOPF ELKIND, A. Nurses, smoking and cancer prevention. International Journal of Health Education 22(2): 92-101, 1979.

(107) KULLANDER, S., KALLEN, B. A prospective study of smoking and pregnancy. Acta Obstetrica Gynecologica Scandinavica 50(1), 83-94, 1971.

(108) KUZMA, J. W., PHILLIPS, R. L. Characteristics of women who discontinued smoking during pregnancy—a preliminary report. September 1979.

(109) LA FARGE, P. An uptight adolescence. Daedalus 100(4): 1159-1175, Fall 1971.

(110) LAMPMAN, J. H. Women hospital workers' smoke. (Letter). New England Journal of Medicine 299(15): 836-837, October 1978.

(111) LANESE, R. R., BANKS, F. R., KELLER, M. D. Smoking behavior in a teenage population: a multi-variate conceptual approach. American Journal of Public Health 62(6): 807-813, June 1972.

(112) LARSON, P. S., SILVETTE, H. Tobacco—Experimental and Clinical Studies. Supplemental III. Baltimore, Williams and Wilkins Company, 1975, 798 pp.

(113) LEVITT, E. E., EDWARDS, J. A. A multivariate study of correlative factors in youthful cigarette smoking. Development Psychology 2(1): 5-11, 1970.

(114) LEWIS, C. E., LEWIS, M. A. The potential impact of sexual equality on health. New England Journal of Medicine 297(16): 863-869, October 1977.

(115) LICHTENSTEIN, E., KEUTZER, C. S., HIMES, K. H. "Emotional" role-playing and changes in smoking attitudes and behavior. Psychological Reports 25(2): 379-387, October 1969.

(116) MACCOBY, E. E. Sex Differentiation during Childhood Development. Master Lectures on Developmental Psychology. Washington, D.C., American Psychological Association, 1977.

(117) MACCOBY, E. E., JACKLIN, C. N. The Psychology of Sex Differences. Stanford, California, Stanford University Press, 1974.

(118) MACKIE, M. Lay perception of heart disease in an Alberta community. Canadian Journal of Public Health 64(5): 445-454, September-October 1973.

(119) MANN, L., JANIS, I. L. A follow-up study on the long-term effects of emotional role playing. Journal of Personality and Social Psychology 8(4): 339-342, 1968.

(120) MARLATT, G. A., GORDON, J. R. Determinants of relapse: implications for the maintenance of behavior change. In: Davison, D. (Editor). Behavioral Medicine: Changing Health Lifestyles. New York, Brunner/Mazel, 1979.

(121) MATARRAZZO, J. D., MATARAZZO, R. G. Smoking. International Encyclopedia of Social Science 14: 335-340, 1968.

(122) MAUSNER, B. An ecological view of cigarette smoking. Journal of Abnormal Psychology 81(2): 115-126, 1973.

(123) MAUSNER, B. Report on a smoking clinic. American Psychologist 21: 251-255, 1966.

(124) MAUSNER, J. S., STEPPACHER, R. C. Suicide in professionals: a study of male and female psychologists. American Journal of Epidemiology 98(6): 436-445, 1973.

(125) MCALISTER, A. L., PERRY, C., MACCOBY, N. Adolescent smoking: onset and prevention. Pediatrics 63(4): 650-658, April 1979.

(126) MCKENNELL, A. C., THOMAS, R. K. Adults' and Adolescents' Smoking Habits and Attitudes. Government Social Survey. London, HMSO, 1967, 308 pp.

(127) MERRY, J., PRESTON, G. The effect of buffered lobeline sulphate on cigarette smoking. Practitioner 190: 628-631, May 1963.

(128) MEYER, R. E. Guide to Drug Rehabilitation: A Public Health Approach. Boston, Beacon Press, 1972.

(129) MOSES, F. M. Treating smoking habit by discussion and hypnosis. Diseases of the Nervous System 25(3): 184-188, March 1964.

(130) NATIONAL INSTITUTE OF EDUCATION. Teenage Smoking Immediate and Long Term Patterns. Department of Health, Education, and Welfare, National Institute of Education, November 1979, 259 pp.

(131) NESSELROADE, J. R., BALTES, P. B. Adolescent personality development and historical change: 1970-72. Monographs of the Society for Research in Child Development 39(1, Serial No. 154): 1-80, 1974.

(132) NEWMAN, I. M. Peer pressure hypothesis for adolescent cigarette smoking. School Health Review 1(2): 15-18, 1970.

(133) NEWMAN, I. M. Status of configurations and cigarette smoking in a junior high school. Journal of School Health 40(1): 28-31, 1970.

(134) NISWANDER, K. R., GORDON, M. The women and their pregnancies. (NIH 73-379) Washington, D.C., U.S. Government Printing Office, 1972, 540 pp.

(135) NOLL, C. E. Health professionals and the problems of smoking and health. Report 5. Nurses, behavior, beliefs, and attitudes toward smoking and health. Report on NORC survey 4001. Chicago, University of Chicago, November 1969, 99 pp.

(136) OCHSNER, A., DAMRAU, F. Control of cigarette habit by psychological aversive conditioning: clinical evaluation in 53 smokers. Journal

of the American Geriatrics Society 18(5): 365-369, May 1970.

(137) O'ROURKE, T. W., STONE, D. B. A prospective study of trends in youth smoking. Journal of Drug Education 1(1): 49-61, March 1971.

(138) PALMER, A. B. Some variables contributing to the onset of cigarette smoking among junior high school students. Social Science and Medicine 4: 359-366, 1970.

(139) PEDERSON, L., LEFCOE, N. A psychological and behavioral comparison of ex-smokers and smokers. Journal of Chronic Diseases 29; 431-434, 1976.

(140) PERRI, M. G., RICHARDS, C. S., SCHULTHEIS, K. R. Behavioral self-control and smoking reduction: a study of self-initiated attempts to reduce smoking. Behavior Theropy 8(3): 360-365, June 1977.

(141) PETERSON, D. I., LONERGAN, L. H., HARDINGE, M. G., TEEL, C. W. Results of a stop-smoking program. Archives of Environmental Health 16(2): 211-214, February 1968.

(142) PETO, J. Price and consumption for cigarettes: a case for intervention? British Journal of Preventive and Social Medicine 28: 241-245, 1974.

(143) PFLAUM, J. Smoking behavior: a critical review of research. Journal of Applied Behavioral Science 1: 195-209, 1965.

(144) POMERLEAU, O., ADKINS, D., PERTSCHUK, M. Predictors of outcome and recidivism in smoking cessation treatment. Addictive Behaviors 3: 65-70, 1978.

(145) POPULATION REPORTS. Tobacco—hazards to health and human reproduction. Population Reports Series L(1): 1-39, March 1979.

(146) PYSZKA, R. H., RUGGELS, W. L., JANOWICA, L. M. Health Behavior Change: Smoking Cessation. Menlo Park, California, Stanford Research Institute, December 1973, 31 pp.

(147) RAW, M. The treatment of cigarette dependence. In: Israel, Y., Glaser, F. B., Kalant, H., Popham, R. E., Schmidt, W., Smart, R. G. (Editors). Research Advances in Alcohol and Drug Problems, Volume 4. New York, Plenum Press, 1978, pp. 441-485.

(148) REEDER, L. G. Sociocultural factors in the etiology of smoking behavior: an assessment. In: Jarvik, M. E., Cullen, J. W., Gritz, E. R., Vogt, T. M., West, L. J. Research on Smoking Behavior. NIDA Research Monograph 17, Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, DHEW Publication No. (ADM) 78-581, 1977, pp. 186-200.

(149) RESNIKOFF, A., SCHAUBLE, P. G., WOODY, R. H. Personality correlates of withdrawal from smoking. The Journal of Psychology 68: 117-120, 1968.

(150) RODE, A., ROSS, R., SHEPHERD, R. J. Smoking withdrawal programme. Personality and cardiorespiratory fitness. Archives of Environmental Health 24(1): 27-36, January 1972.

(151) RODE, A., SHEPHERD, R. J., ROSS, R. Smoking and personality. American Review of Respiratory Diseases 104(6): 929-932, December 1971.

(152) ROSS, C. A. Smoking withdrawal research clinics. In: Zagona, S. V. (Editor). Studies and Issues in Smoking Behavior. Tucson, University of Arizona Press, 1967, pp. 111-130.

(153) ROUSE, B. A., EWING, J. A. Marihuana and other drug use by women college students: associated risk taking and coping activities. American Journal of Psychiatry 130(4): 486-491, April 1973.

(154) ROYAL COLLEGE OF PHYSICIANS. Smoking or Health. London,

Pitman Medical Publishing Company, 1977, 128 pp.

(155) RUDOLPH, J. P., BORLAND, B. L. Factors affecting the incidence and acceptance of cigarette smoking among high school students. Adolescence 11(44): 519-525, Winter 1976.

(156) RUSSELL, M. A. H. Effect of electric aversion on cigarette smoking. British Medical Journal 1(5688): 82-86, January 1970.

(157) RUSSELL, M. A. H. Tobacco dependence: is nicotine rewarding or aversive? In: Krasnegor, N. A. (Editor). Cigarette Smoking as a Dependence Process. National Institute on Drug Abuse Monograph 23. Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, January 1979, pp. 100-122.

(158) RUSSELL, M. A. H. Tobacco smoking and nicotine dependence. In: Gibbons, R. J., Israel, Y., Kalant, H., Popham, R. E., Schmidt, W., Smart, R. G. (Editors). Research Advances in Alcohol and Drug Problems, Volume 3. New York, John Wiley and Sons, 1976, pp. 1-47.

(159) RUSSELL, M. A. H., ARMSTRONG, E., PATEL, U. A. Temporal contiguity in electric aversion therapy for cigarette smoking. Behaviour Research and Therapy 14(2): 103-123, 1976.

(160) RUSSELL, M. A. H., PETO, J., PATEL, U. A. The classification of smoking by factorial structure of motives. The Journal of the Royal Statistical Society, Series A (General) 137(Part 3): 313-346, 1974.

(161) SALBER, E. J., ARELIN, T. Smoking behavior of Newton school children—5 year follow-up. Pediatrics 40(3, Part I): 363-372, September 1967.

(162) SALBER, E. J., WELSH, B., TAYLOR, S. V. Reasons for smoking given by secondary school children. Journal of Health and Human Behavior 4: 118-129, 1963.

(163) SCHACTER, S. Regulation on withdrawal and nicotine addiction. In: Krasnegor, N.A. (Editor). Cigarette Smoking as a Dependence Process. NIDA Monograph No. 23. Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, January 1979, pp. 123-133.

(164) SCHAUBLE, P. G., WOODY, R. H., RESNIKOFF, A. Educational therapy and withdrawal from smoking. Journal of Clinical Psychology 23: 518-519, 1967.

(165) SCHIEVELBEIN, H., HEINEMANN, G., LOSCHENKOHL, K., TROLL, C., SCHLEGEL, J. Metabolic aspects of smoking behaviour. In: Thornton, R. E. (Editor). Smoking Behaviour. Edinburgh, Churchill Livingstone, 1978, pp. 371-390.

(166) SCHNEIDER, F. W., VANMASTRIGT, L. A. Adolescent-preadolescent differences in beliefs about smoking. Journal of Psychology 87(First Half): 71-81, May 1974.

(167) SCHULZ, W., SEEHOFER, F. Smoking behaviour in Germany—the analysis of cigarette butts (KIPA). In: Thornton, R. E. (Editor). Smoking Behaviour, Edinburgh, Churchill Livingstone, 1978, pp. 259-276.

(168) SCHUMAN, L. Patterns of smoking behaviour. In: Jarvik, M. E., Cullen, J. W., Gritz, E. R., Vogt, T. M., West, L. J. (Editors). Research on Smoking Behavior. NIDA Research Monograph No. 17. Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, DHEW Publication No. (ADM) 78-581, December 1977, pp. 36-66.

(169) SCHWARTZ, J. L. Smoking cures: ways to kick an unhealthy habit. In: Jarvik, M. E., Cullen, J. W., Gritz, E. R., Vogt, T. M., West, L. J. (Editors). Research on Smoking Behaviour. National Institute on Drug Abuse, Monograph No. 17, DHEW Publication No. (ADM) 78-581, December 1977, pp. 308-338.

(170) SCHWARTZ, J. D., DUBITSKY, M. One-year follow-up results of a smoking cessation program. Canadian Journal of Public Health 59: 161-165, 1968.

(171) SCHWARTZ, D., GOUJARD, J., KAMINSKI, M., RUMEAU-ROUQUETTE, C. Smoking and pregnancy. Results of a prospective study of 6,989 women. Revue Europeene d'Etudes Cliniques et Biologiques 17(9): 867-879, 1972.

(172) SHEKELLE, R. B., SCHOENBERGER, J. A., STAMLER, J. Correlates of the JAS Type A behavior pattern score. Journal of Chronic Diseases 29(6): 381-394, June 1976.

(173) SHIFFMAN, S. M. The tobacco withdrawal syndrome. In: Krasnegor, N.A. (Editor). Cigarette Smoking as a Dependence Process. NIDA Research Monograph 23, Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse, January 1979, pp. 158-185.

(174) SIMON, W. E., PRIMAVERA, L. H. The personality of the cigarette smoker: some empirical data. International Journal of the Addictions 11 (1): 81-94, 1976.

(175) SIMS, E. A. H. Experimental obesity, dietary-induced thermogenesis and their clinical implications. Clinics in Endocrinology and Metabolism 5(2): 377-395, July 1976.

(176) SMITH, G. M. Relations between personality and smoking behavior in pre-adult subjects. Journal of Consulting and Clinical Psychology 33(6): 710-715, 1969.

(177) SMITH, G. M., FOGG, C. P. Psychological predictors of early use, late use, and nonuse of marihuana among teenage students. In: Kandel, D. B. Longitudinal Research on Drug Use: Empirical Findings and Methodological Issues. Washington, D.C., Hemisphere Publishing Corp., 1978, pp. 101-113.

(178) SMITH, R. C. The magazines' smoking habit. Columbia Journalism Review 16(5): 29-31, February 1978.

(179) SOFFER, A. Discussion of physicians' attitudes toward smoking. Diseases of the Chest 54(3): 182-185, September 1968.

(180) SROLE, L., FISCHER, A. K. The social epidemiology of smoking behavior 1953 and 1970: the midtown Manhattan study. Social Science and Medicine 7: 341-358, 1973.

(181) STEFFENHAGEN, R. A., MCAREE, C. P., NIXON, H. L. Drug use among college females: socio-demographic and social psychological correlates. The International Journal of the Addictions 7(2): 285-303, 1972.

(182) STEPPACHER, R. C., MAUSNER, J. S. Suicide in male and female physicians. Journal of the American Medical Association 228(13): 323-328, April 1974.

(183) STERLING, T. D., WEINKAM, J. J. Smoking characteristics by type of employment. Journal of Occupational Medicine 18(11): 743-754, November 1976.

(184) STEWART, A. L., BROOK, R. H., KANE, R. L. Conceptualization and measurement of health habits for adults in the Health Insurance Study: Volume 1, Smoking. Department of Health, Education, and

Welfare, June 1979, 62 pp.

(185) STRELTZER, N. E., KOCH, G. V. Influence of emotional role-playing on smoking habits and attitudes. Psychological Reports 22(3-1): 817-820, June 1968.

(186) SUEDFELD, P., IKARD, F. F. Attitude manipulation in restricted environments: IV. Psychologically addicted smokers treated in sensory deprivation. British Journal of Addictions 68(2): 170-176, 1973.

(187) TAMERIN, J. S. The psychodynamics of quitting smoking in a group. American Journal of Psychiatry 129(5): 101-107, November 1972.

(188) THOMPSON, E. L. Smoking education programs 1960-1976. American Journal of Public Health 68(3): 250-257, March 1978.

(189) TONGAS, P., GOODKIND, S., PATTERSON, J. An investigation of effects of post-treatment maintenance on the cessation of smoking within four behavioral treatment modalities. Paper presented at the Western Psychological Association Convention, Los Angeles, 1976.

(190) TRAHAIR, R. C. S. Giving up cigarettes: 222 case studies. Medical Journal of Australia 1: 929-932, May 1967.

(191) TURLE, G. C. An investigation into the therapeutic action of hydroxyzine (Atarax) in the treatment of nervous disorders and the control of the tobacco-habit. Journal of Mental Science 104: 826-833, July 1958.

(192) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE. Use of Tobacco. Practices, Attitudes, Knowledge, and Beliefs, United States—Fall 1964 and Spring 1966. Department of Health, Education, and Welfare, National Clearinghouse for Smoking and Health, July 1969, 807 pp.

(193) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE. Adult Use of Tobacco 1970. Department of Health, Education, and Welfare, National Clearinghouse for Smoking and Health, DHEW Publication No. (HSM) 73-8727, June 1973.

(194) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE. Adult Use of Tobacco 1975. Department of Health, Education, and Welfare, National Clearinghouse for Smoking and Health, CDC 21-74-520, June 1976.

(195) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE. Survey of Health Professionals, 1975. Department of Health, Education, and Welfare, National Clearinghouse for Smoking and Health, CDC 21-74-552 (P), June 1976.

(196) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE. 1975 Study of Cigarette Smoking Among Four Health Professional Groups in the United States: Basic Tabulations. Department of Health, Education, and Welfare, Center for Disease Control, Bureau of Health Education, National Clearinghouse for Smoking and Health, September 1976.

(197) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE. Teenage Smoking: National Patterns of Cigarette Smoking Ages 12 through 18 in 1972 and 1974. Department of Health, Education, and Welfare, National Clearinghouse for Smoking and Health, DHEW Publication No. (NIH) 76-931.

(198) U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE. Smoking and Health: A Report of the Surgeon General. Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, DHEW Publication No. (PHS) 79-50066, January 1979, 1251 pp.

(199) U.S. PUBLIC HEALTH SERVICE. Smoking Behavior and Attitudes:

Physicians, Dentists, Nurses, Pharmacists. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Clearinghouse for Smoking and Health, 1977.

(200) VUORI, H., HIMANEN, P., JANNINEN, J., JARVINEN, M., RANTANEN, T. The smoking habits of Finnish physicians. International Journal of Health Education 14(2): 68-74, 1971.

(201) WARNECKE, R. B., ROSENTHAL, S., GRAHAM, S., MANFREDI, C. Social and psychological correlates of smoking behavior among black women. Journal of Health and Social Behavior 19: 397-410, December 1978.

(202) WARNER, K. The effects of the anti-smoking campaign on cigarette consumption. American Journal of Public Health 67(7): 645-650, July 1977.

(203) WATERS, W. E. Smoking and neuroticism. British Journal of Preventive and Social Medicine 25: 162-164, 1971.

(204) WEST, D. W., GRAHAM, S., SWANSON, M., WILKINSON, G. Five year follow-up of a smoking withdrawal clinic population. American Journal of Public Health 67(6): 536-544, June 1977.

(205) WESTLING-WIKSTRAND, H., MONK, M. A., THOMAS, C. B. Some characteristics related to the career status of women physicians. Johns Hopkins Medical Journal 127(5): 213-286, November 1970.

(206) WETTERQVIST, H. Experimental work in the antidotal treatment of smokers, 1966-1967. Lakartidningen 70: 3591-3595, 1973.

(207) WETTERQVIST, H. Points in the matter of giving up smoking. Smoking withdrawal in Lund. Social-Medicinsk Tidskrift 2: 111-112, 1971.

(208) WHITEHEAD, R. W., DAVIES, J. M. A study of methylphenidate and diazepam as possible smoking deterrents. Current Therapy and Research 6(5): 363-367, May 1964.

(209) WILHELMSEN, L. One year's experience in an antismoking clinic. Scandinavian Journal of Respiratory Diseases 49(4): 251-259, 1968.

(210) WILHELMSEN, L., FAITHELL, P. New study on the smoking habits of Swedish physicians. UICC Technical Report Series 11: 66-67, 1974.

(211) WILLIAMS, J. H. Psychology of Women. New York, W. W. Norton & Co., 1979, 506 pp.

(212) WILLIAMS, T. M. Summary and Implications of Review of Literature Related to Adolescent Smoking. Department of Health, Education, and Welfare, Health Services and Mental Health Administration, 1971, 59 pp.

(213) WOHLFORD, P. Initiation of cigarette smoking: is it related to parental smoking behavior? Journal of Consulting and Clinical Psychology 34(2): 148-151, 1970.

(214) WORDEN, J. K., SWEENEY, R. R., WALLER, J. A. Audience interest in mass media messages about lung disease in Vermont. American Journal of Public Health 68(4): 378-382, April 1978.

(215) WYNDER, E. L., KAUFMAN, P. L., LESSER, R. L. A short-term follow-up study on ex-cigarette smokers, with special emphasis on persistent cough and weight gain. American Review of Respiratory Diseases 96(4): 645-655, October 1967.

(216) YANKELOVICH, SKELLY, AND WHITE, INC. A Study of Cigarette Smoking among Teen-age Girls and Young Women. Summary of the Findings. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute, DHEW Publication No. (NIH) 77-1203, 1977.

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